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AIR POLLUTION: AIR QUALITY CRITERIA FOR PARTICULATE MATTER

NATO Committee on the Challenges of Modern Society

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AIR QUALITY CRITERIA
FOR
PARTICULATE MATTER

A Report By CCMS/NATO Expert Panel
On
Air Quality Criteria

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Dr. Jean Ternisien, The Republic of France

November 1971

FOREWORD

The material presented in this document represents a first attempt by the Expert Panel for Air Quality Criteria, Committee on Challenges to Modern Society, NATO, to compile a compendium of information using the United States Air Quality Criteria for Particulate Matter as a basic document on the effects of particulate matter in the ambient air on man, materials, vegetation, and weather. Definitive, quantitative relationships between concentration of particulate matter and consequent effects are not well established. In years past, concern over these detrimental effects has been limited, although some notable scientific investigations were undertaken. Recent interest and activity on the part of the public, governmental, scientific, academic, and industrial sectors of our communities have stimulated research on the effects of pollution on man and his environment. The result is an ever-increasing body of knowledge that must be analized, interpreted, and finally expressed as information supporting or refuting suggested criteria. The assimilation of this new knowledge is not a task that can be undertaken by a small body of men--such as a committee of this nature. Rather, the responsibility for assuring that criteria continually reflect the most significant information available rests with those same sectors of our communities that have inspired all of us to strive to improve our environment--the public, the government, the scientific, the academic, and the industrial sectors.

The Panel encourages and welcomes new data, comments, and constructive criticism of this document. We suggest that a procedure be established whereby this document and documents to follow can be revised and updated periodically.

For the Expert Panel,
R. E. Engel, Ph. D., Chairman

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The working panel which worked diligently through the course of the year receives the highest praise. The panel members were:

Pilot Countries

Dr. F. Gordon Hueter, USA
Dr. H. Wilfried Goerke, FRG
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Lastly, acknowledgement must be given to those secretaries and other personnel who have made the panel effort possible.

Ronald E. Engel, Ph.D.
Panel Chairman

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Chapter 1

ATMOSPHERIC PARTICLES: DEFINITIONS, PHYSICAL PROPERTIES, SOURCES, AND CONCENTRATIONS

A. INTRODUCTION

Atmospheric particles are chemically a most diverse class of substances; they do, however, have a number of physical properties in common, and for this reason are generally classed in a single category, sometimes referred to as aerosols.

Some workers restrict the concept of particulate matter to the solid phase; this distinction is, however, difficult to make in practice and is probably not proper. Others refer to airborne particles as nuclei because some particles have a role in the nucleation of condensation, especially of liquid or solid water. In this document, the term "particle" is used to mean any dispersed matter, solid or liquid, in which the individual aggregates are larger than a single small molecule (about 0.0002 micrometer in diameter), but smaller than about 500 micrometers (µm's). The present document discusses the several classes of particles by specifying the size or size interval in micrometers and, where appropriate, the physical state. Figure 1-1 provides a graphic scheme for relating meteorologic nomenclature for aerosols to the particle sizes.[1] In this document, "size" ordinarily refers to particle diameter or Stokes' diameter. Settling velocities for various Stokes' diameters are shown in Figure 1-2.

If the density of the particles is not known, it may arbitrarily be assigned a value of 1 g/cm^3 ; in this case, the diameter will be referred to as the "aerodynamic diameter"--that is, the diameter of a

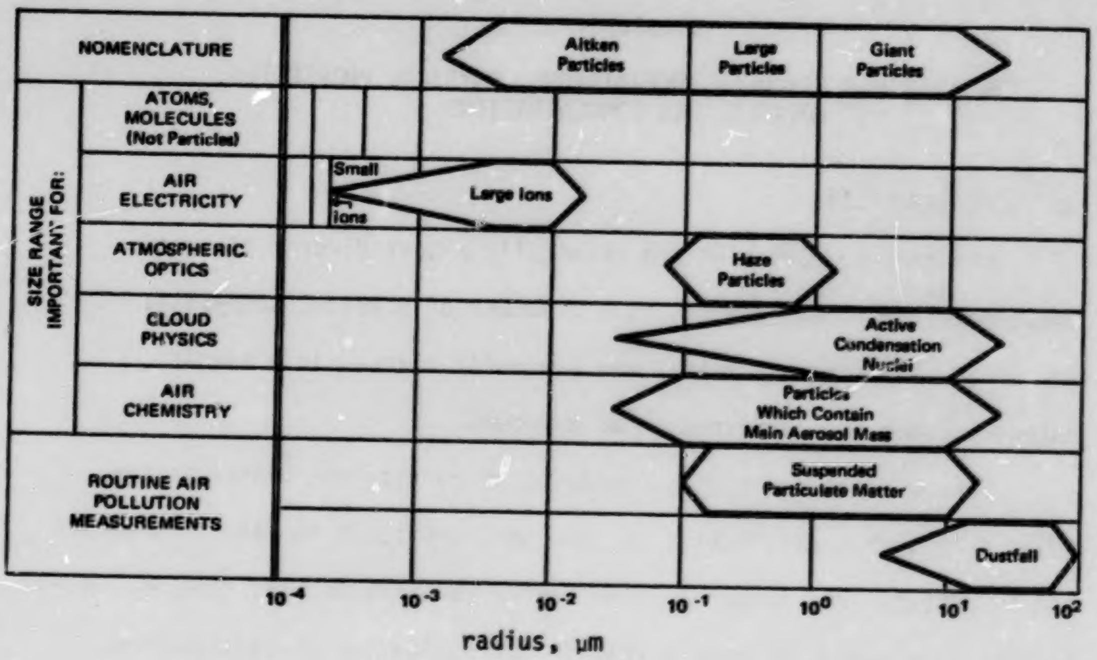

Figure 1-1. Sizes of atmospheric particulate matter.[1] (The figure shows the ranges of particle size (radius) of various types of particulate matter which are found in the earth's atmosphere.)

spherical particle of unit density has the same terminal fall velocity in still air as the particles in question.

Some of the smallest particles may be no more than statistical aggregations of gas molecules which act as a particle at one instant and cease to exist at the next. Solid particles and liquid droplets may be formed in the atmosphere by condensation of a vapor. Solid particles produced by abrasion or grinding are not spherical and are called dust.

This discussion of size classes must not obscure the fact that there is a continuous spectrum of sizes among the particles in the atmosphere and a corresponding continuous gradation of all their size-dependent properties. The distribution of particle sizes usually encountered approximates closely a log-normal distribution. Figure 1-3

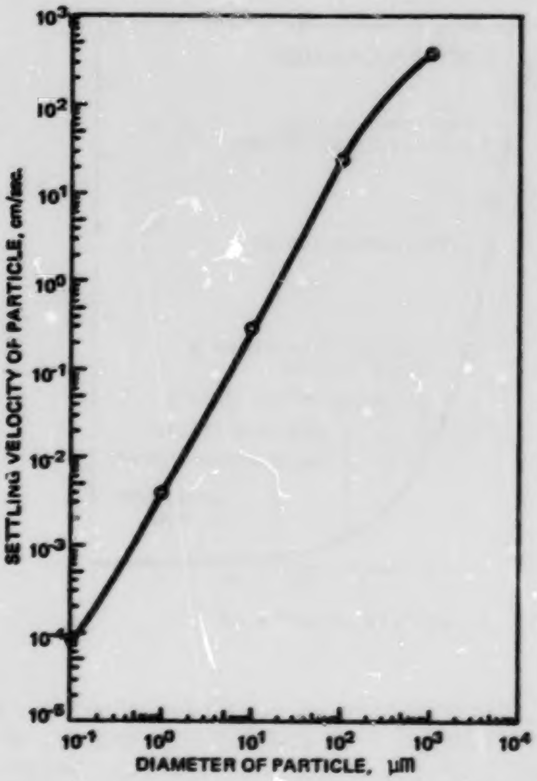

Figure 1-2. Settling velocities in still air at 0° C and 760 mm pressure for particles having a density of 1 g/cm^3 as a function of particle diameter. (This graph shows that, for spherical particles of unit density suspended in air near sea level, Stokes' law applies over a considerable range of particle sizes, where the line is straight, but that correction is required at the extremes where the line begins to curve.)

and 1-4 show the linear and logarithmic frequency distribution curves for the log-normal distribution. When plotted using log-probability scales, cumulative log-normal distribution is a straight line. Such a plot is shown in Figure 1-5, and the experimental points give an idea of the extent to which the size distribution of a typical industrial dust approximates log-normal.

A log-normal distribution curve can be specified in terms of (1) the most probable size, which in this distribution is identical with the

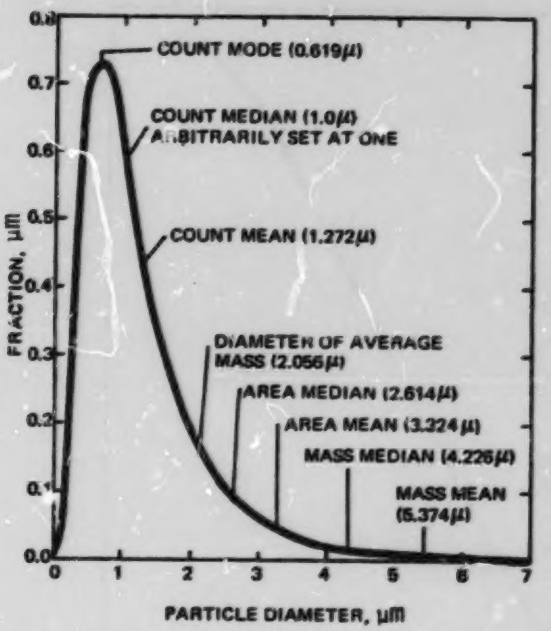

Figure 1-3 Log-normal distribution of particles showing various average diameters.[2] (The graph is drawn from probability theory, assuming a count median diameter of 1 μm and shows the numerical values relative to that diameter of several other weighted average diameters discussed in the text.)

geometric mean, M_g, and (2) the geometric standard deviation, σ_g. It can be shown that the 50-percent point of the cumulative graph (Figure 1-5) corresponds to M_g, while the ratio of the 84.1-percent point to the 50-percent point is σ_g.

Several averages of particle size may be used; these may be based on averages of numbers, areas, masses, etc. Some of the most important averages are indicated in Figure 1-3, and the numerical values calculated for the distribution are given relative to a count (number) median diameter of 1 μm.[2] More extensive mathematical treatments of particle statistics are given in books by Cadle[3] and by Drinker and Hatch.[4]

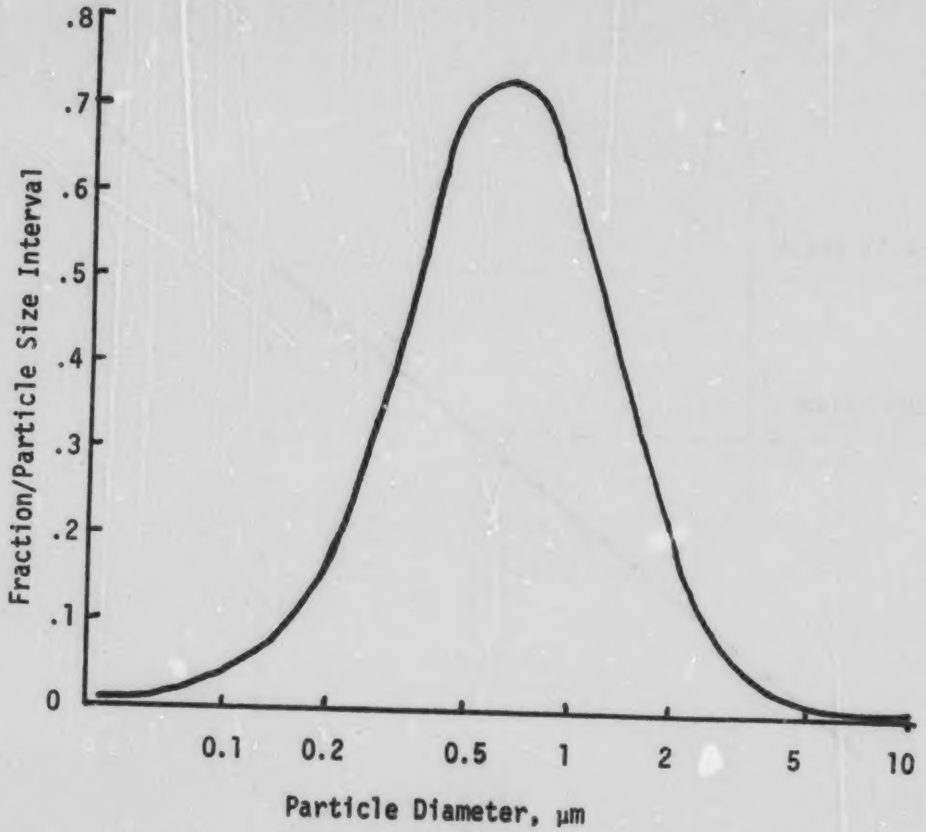

Figure 1-4. Particle size distribution of Figure 1-3, plotted logarithmically. (If the particle size distribution is log-normal, the graph is symmetrical when plotted logarithmically. The figure should be contrasted with Figure 1-3.)

B. PROPERTIES OF ATMOSPHERIC PARTICULATE MATTER

Since particulate matter may consist of such a wide variety of substances, a discussion of chemical properties of the class cannot be specific. Some selected chemical properties of important individual species will be discussed later in connection with analytical methods. Biological properties are most conveniently grouped in terms of the effects produced.

Physical properties are equally diverse. However, three general classes of physical properties can reasonably be said to apply to all particulate matter. These properties all involve the interface between the particle and its surroundings; they are (1) surface properties,

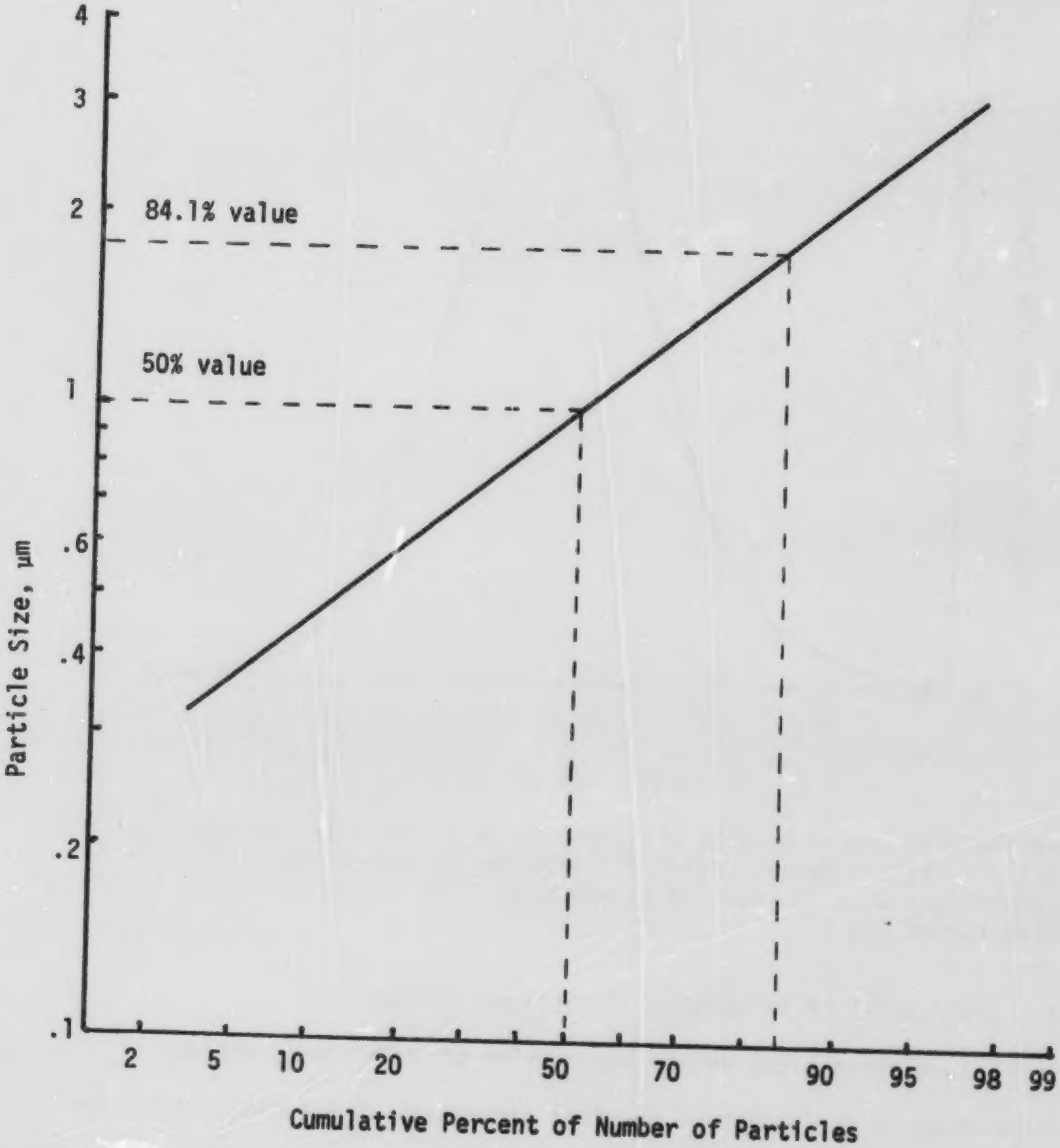

Figure 1-5. Cumulative log-probability curve for the distribution of Figure 1-4. (In a cumulative plot, the experimental points fall on a straight line when the size distribution is log-normal. The 50% point corresponds to the geometric mean, M_g (1.0 µm in this case), and the ratio of the 84.1% point to this (about 1.8/1.0 or 1.8, in this case) is the geometric standard deviation, σ_g. In practice, the experimental points usually lie near a straight line, as shown.)

(2) motions, and (3) optical properties. A discussion of the properties of aerosols has been given by Corn,[1] and it should be used to extend the remarks offered below.

1. Surface Properties

Surface properties include sorption, nucleation, adhesion, etc. Sorptive behavior can best be understood by considering the impact of individual molecules on the particle surface. If this impact is perfectly elastic and rebound is truly instantaneous, nothing happens. If, however, rebound is delayed or the velocity of rebound is smaller than that of impact, then there will be a local accumulation of the gas on or near the particle surface. If the delay is great, a substantial fraction of the surface may be covered--a phenomenon known as adsorption. If the delay is caused specifically by a chemical interaction between the surface and the gas, the process is known as chemisorption; absorption refers to the situation in which the gas is dissolved into the particle.

A vapor (i.e., a gas below its critical temperature), present in amounts comparable to its equilibrium vapor pressure, may lead to a deepened sorbed layer, which then takes on the character of a layer of true liquid or solid. If the vapor is supersaturated, a droplet or crystal may grow by further condensation on the sorbed layer. The net result is nucleation, a phenomenon which deserves more consideration. A pure vapor, free of particles, must be highly supersaturated before a condensed phase will form from it, because an energy barrier separates the molecular from the particulate state.

Two like molecules of gas will not generally stick together, and an aggregate of three molecules is still less likely to retain its identity for any length of time. A small aggregate of molecules is therefore unstable. On the other hand, if a particle is split in two, energy is required to create the new surfaces, since the combined surface area of the two fragments is greater than that of the original particle (surface energy increases with a decrease in size). At some point, these two trends of decreasing stability meet at a maximum which corresponds to a certain particle size. If a molecular aggregate can reach this size, then the addition of a single molecule puts it over the energy barrier and it will become more stable by collecting still more molecules. Conversely, the loss of a single molecule from a nucleus of critical size can destroy its stability with the probable result that it will return to the molecular or gaseous state.

The important point is that the critical particle generally contains some tens of molecules which must all come together at once. Unless the vapor concentration is high, this is an improbable event; for some substances, homogeneous nucleation may require supersaturations of many hundredfold. However, a complete sorbed layer on a particle surface behaves like a drop of the same diameter as the particle, and the energy barrier to producing a droplet is avoided. Since particles are always present in the atmosphere, nucleation on them is of widespread occurrence.

The last of the surface properties of consequence is adhesion. All available evidence suggests that solid particles with diameters less than 1 μm (and liquid particles regardless of size) always adhere when they collide with each other or with a larger surface. Other factors being equal, reentrainment or rebound becomes increasingly probable with increasing particle size. Alternatively, the adhesive property can be considered in terms of the surface energy of small particles or in terms of the more complex shear forces acting to dislodge the larger particles.

2. Motion

The second major class of properties common to all particles, regardless of composition, is the mode of motion. Particles with diameters less than 0.1 μm undergo large random (Brownian) motions caused by collision with individual molecules. Particles larger than 1 μm have significant settling velocities, and their motions can vary significantly from the motion of the air in which they are borne. For particles between 0.1 and 1 μm, settling velocities in still air, though finite, are small compared with air motions. Despite fairly high concentrations, coagulation is somewhat slower than for particles smaller than 0.1 μm because of decreased Brownian motion. Nevertheless, the operation of this mechanism, together with the processes which generate larger particles and which remove particles from the air, causes the whole population of particulate matter in the air to tend towards a constant size distribution.

Although actual settling times in the atmosphere tend to differ from those computed from Stokes' Law, because turbulence tends to offset gravitational fall, the particles larger than 5 or 10 μm to a large extent are removed by gravity and other inertial processes. The size distribution of a freshly generated aerosol may contain particles as large as 500 μm. These large particles settle rapidly. It is not until the size range of approximately 5-10 μm is considered that significant time periods are required to effect elimination from the distribution.

3. Optical Properties

The final class of physical properties to be discussed is the effects of particles on light. This is clearly of importance in effects on visibility, and it is through optical effects that particles usually are perceived in the atmosphere. Once again, particles in the size range

0.1 to 1 μm exhibit properties showing a transition between two extreme cases.

Particles below 0.1 μm are sufficiently small compared to the wavelength of light to obey approximately the same laws of light scattering as do molecules. This so-called Rayleigh scattering varies as the sixth power of the particle diameter and is fairly inconsequential in its effects on visibility. On the other hand, particles very much larger than 1μm are so much larger than the wavelength of visible light that they obey the same laws as macroscopic objects, intercepting or scattering light roughly in proportion to their cross-sectional area. Particles in the intermediate size range obey complex scattering laws set forth by Mie;[5] these laws are beyond the scope of this discussion. Because the particle dimensions are of the same order of magnitude as the wavelength of visible radiation, interference phenomena play a complicating role, and a given scattering behavior may correspond to several particle sizes. Unfortunately, this is precisely the size range which is most effective in light scattering and thus the one most needful of study. A more complete discussion of optical effects is in Chapter 3.

C. CHEMICAL REACTIONS OF ATMOSPHERIC PARTICULATE MATTER

In view of the diverse chemical compositions of particles, it is not possible to make general statements about the chemical reactions of particulate atmospheric pollutants. The following discussion refers to some specific reaction systems that have been studied. Both particle-gas and particle-particle reactions can occur, but the latter class has been studied to an even lesser extent than the former. Such particle-particle reactions should certainly occur in the size range below 0.1 μm, where collision between particles is frequent, but among particles large enough to be readily studied, collisions are relatively infrequent in the atmosphere because of low concentrations. Samples of particles collected on filters may, however, react and subsequent analysis can be very misleading if this fact is not taken into account.

One of the particle-gas systems, the reaction between sulfuric acid mist and ammonia gas, was investigated by Robbins and Cadle.[6] At high humidities the reaction rate was limited by diffusion of ammonia to the mist droplets. At low humidities the droplets were viscous enough for diffusion of the reaction product away from the surface of the drops to be the rate-determining step. This work shows the effect of accumulation of reaction products and it attempts to explain the role of humidity in a gas-particle reaction.

Goetz and Pueschel[7] have fully revealed the complexity of even a simplified model of the photochemical air pollution found in Los Angeles. The one clear relationship is that the amount of reaction product deposited on nuclei supplied from the gas phase is proportional to the surface area of the nuclei. The humidity effect is complex and depends upon the amounts and the order of addition of the other substances present. The obvious reactants (olefins, nitrogen dioxide, and sulfur dioxide) differ in action as well. Amounts of sulfur dioxide of the order of 5 $\mu g/m^3$ (2 ppm) depress aerosol formation, while larger amounts 40 $\mu g/m^3$ (15 ppm) increase it. Nitrogen dioxide is more effective if mixed with nuclei before mixing with the olefin.

Interactions between sulfur dioxide and metal oxide aerosols have been studied by Smith et al.[8] at ambient conditions of temperature and humidity. In measurements that included an adsorption isotherm for sulfur dioxide on dispersed particles, preferential chemisorption on iron oxide and aluminum oxide aerosols was observed at low sulfur dioxide concentrations up to 5 $\mu g/m^3$ followed by multilayered physical adsorption at higher concentrations.

D. SOURCES OF ATMOSPHERIC PARTICULATE MATTER

In a broad sense, particles in the atmosphere are produced by two mechanisms: those in the size range below 1 μm arise principally by condensation, while larger particles result from comminution, although there is considerable overlap. For example, Preining et al.[9] showed the presence of many particles smaller than 1 μm in the spray from a nebulizer, while the formation of very small drops during the rupture of bubbles has been demonstrated.[10] Dry-grinding processes are rarely efficient in producing particle sizes below a few micrometers because of the rapid increase in energy necessary to produce the additional surface.

Combustion is complex in that it may produce four distinct types of particles. These may arise in the following ways:

1. The heat may vaporize material which subsequently condenses to yield particles in the size range between 0.1 and 1 μm.
2. The energy may produce particles of very small size, below 0.1 μm; these particles may be of short life as a result of being unstable molecular clusters.
3. Mechanical processes may reduce either fuel or ash to particles sized larger than 1 μm; these may be entrained in the combustion gases.
4. If the fuel itself is an aerosol during combustion, a very fine ash may escape directly.

5. Partial combustion of fossil fuels may produce soot.

Particles larger than 10 μm frequently result from mechanical processes such as wind erosion, grinding, spraying, etc.

The sources of dust are usually apparent. For example, a dustfall sample nearly always contains particles of local soil. Another large fraction will be materials dropped on the ground and pulverized by vehicles, pedestrians, or wind action. Although actual soot flocs are increasingly absent as better home heating is used, there may be partially burnt trash from inefficient incinerators. Finally, the process dust that is characteristic of local industry may be present. In urban locations, particles between 1 and 10 μm generally reflect industrial and combustion processes with some local soil also present, but in maritime locations, the bulk of the airborne sea salt will be found in particles of this size. In short, atmospheric particles in the 1 to 10 μm range tend to have a composition that is characteristic of local sources and soils.

As mentioned before, it is difficult to form small particles by size reduction. Therefore, the class of particles between 0.1 to 1 μm, compared with the larger particles, tends to contain larger amounts of condensation products. Products of combustion predominate together with photochemically produced aerosols. Particles below 0.1 μm have not been characterized chemically, but the increase over the natural level that is characteristic of cities seems to be entirely the result of combustion.

Table 1-1 shows typical a[illegible]missions of particles from various types of sources in St. Louis, Missouri, United States (population 2,331,000) and in Ankara, Turkey (population 906,000).[11] Identical procedures were used in collecting these two sets of data.

Table 1-1. 1969 EMISSION INVENTORY OF PARTICULATE MATERIAL, METRIC TONS PER YEAR[11]

Source Category	City	
	St. Louis, United States	Ankara, Turkey
Transportation		
Motor vehicles	5,220	2,090
Other	2,760	240
Subtotal	7,980	2,330
Stationary Fuel Combustion		
Industry	60,900	4,870
Residential	2,760	7,470
Commercial, institutional Governmental	4,940	4,580
Electric generation	49,400	1,210
Subtotal	118,000	18,130
Refuse Disposal		
Incineration	2,180	n
Open burning	3,140	1,200
Subtotal	5,320	1,200
Industrial Processes	37,500	3,210
Evaporative Losses	--	--
GRAND TOTAL	168,800	24,870

n = negligible

E. ATMOSPHERIC PARTICULATE MATTER IN URBAN AREAS

1. Suspended Particulate Matter

The bulk of current data on suspended particles generally does not discriminate on the basis of size. Most of the data (e.g., from NASN, the U. S. National Air Surveillance Network) are obtained from high-volume filter samples.[12,13]

Clark and Whitby[14] have shown the feasibility of estimating particle size distributions from the weight of high-volume filter samples. Blifford and Meeker[15] have applied factor analysis to quantitative determinations of the chemical composition of high-volume filter samples to provide an estimate of the relative contribution by classes of sources to suspended particles in several cities.

Some measurements of particle size distributions are now being made routinely, using cascade impactors.[16] Table 1-2 shows results of these measurements for several cities in the United States.[17] These data indicate that a large percentage of the mass of airborne particles in urban atmospheres is accounted for by particles less than 1 μm in diameter. (The significance of these small diameter particles is discussed in Chapter 9.)

Table 1-3 lists the average suspended particle concentrations for a number of cities.[17] For the most part, measurements were taken at a single sampling station in the downtown area of the city.

In urban areas, average suspended particle mass concentrations range from 60 to 220 $\mu g/m^3$, depending on the size of the city and on its industrial activity. In heavily polluted areas, particle concentrations as high as 2,000 $\mu g/m^3$ have been measured over a 24-hour sampling period. In contrast, average suspended particle mass concentrations range from about 10 $\mu g/m^3$ in remote nonurban areas to about 60 $\mu g/m^3$ in near urban areas.

Based on analyses of 10 years of U.S. data at approximately 370 sites, the highest seasonal average exceeds the annual mean by 15 to 20 percent. Seasonal averages for the highest 2 percent of the sites exceeded

Table 1-2. SIZE DISTRIBUTION OF SUSPENDED PARTICLES COLLECTED IN SEVERAL UNITED STATES CITIES, 1970[17]

City	Number of Samples	Average Concentration μg/m^3	Average Percent of Particle Mass Having Diameter:	
			≤1μm	≤2μm
Chicago, Illinois	21	86	55	68
Cincinnati, Ohio	18	74	59	74
St. Louis, Missouri	22	73	54	68
Denver, Colorado	21	59	65	75
Philadelphia, Pennsylvania	20	58	67	80
Washington, D. C.	23	56	68	81

the annual mean by 50 percent and for the lowest 2 percent by about 5 percent. Individual 24-hour maximum sample concentrations vary widely from the annual mean, and on the average this variation is from 280 to 300 percent. Variations as high as 700 percent of the annual mean are found for the high 2 percent of the samples. Sunday and holiday data are usually 15 to 20 percent below weekday concentrations.

Particle concentrations in air have both diurnal and annual (seasonal) cycles which for most cities are generally predictable in shape. A city with cold winters will experience a seasonal maximum in midwinter as a result of increased fuel use for space heating. A daily maximum in the morning, probably between 6 and 8 o'clock, usually relates to a combination

Table 1-3. AVERAGE SUSPENDED PARTICLE CONCENTRATIONS FOR SELECTED UNITED STATES URBAN AREAS, μg/m^3 [17]

City	Period of Record	Geometric Mean Concentration μg/m^3
Seattle, Washington	1960-1969	66
New Orleans, Louisiana	1963-1969	83
Washington, D. C.	1960-1969	92
Boston, Massachusetts	1960-1962; 1964; 1966; 1968; 1969	114
Denver, Colorado	1960-1969	121
Los Angeles, California	1960-1969	125
Chicago, Illinois	1961-1969	128
Detroit, Michigan	1960-1969	131
New York, New York	1960-1969	153
Phoenix, Arizona	1961-1969	157

of meteorological factors and an increase in the strength of sources of particulates, including the automobile traffic.

A reflection of the effect of the strength of various sources can also be seen in the previously mentioned lower weekend and holiday versus weekday concentrations.

Particles of various size ranges show different patterns of concentration in their daily cycles in regions where photochemical pollution predominates. Thus, the highest concentration (number density) of particles less than 0.1 μm in size will occur between sunrise and midday, while particles between 0.1 and 1.0 μm will be most numerous around noontime--after the sun has had an opportunity to cause photochemical reactions. Particles larger than 1.0 μm may show no trend in concentration.

The above concentrations generally relate to samples taken in the center-city commercial district. This portion of the community will generally not show annual average concentrations as high as those found in various industrial areas; however, they are among the higher area concentrations in a community. Annual concentrations in outlying residential areas generally will be about one-half of that found in the city center.

Table 1-4 gives concentrations of certain specific contaminants found in total suspended particulate matter.[12]

Table 1-4. ARITHMETIC MEAN AND MAXIMUM URBAN PARTICULATE CONCENTRATIONS IN THE UNITED STATES, BIWEEKLY SAMPLINGS, 1964 to 1965[12]

Pollutant	Number of stations	Concentrations μg/m³	
		Arith. average	Maximum
Suspended particulates	291	105	1254
Fractions:			
Benzene-soluble organics	218	6.8	(a)
Nitrates	96	2.6	39.7
Sulfates	96	10.6	101.2
Ammonium	56	1.3	75.5
Antimony	35	0.001	0.160
Arsenic	133	0.02	(a)
Beryllium	100	<0.0005	0.010
Bismuth	35	<0.0005	0.064
Cadmium	35	0.002	0.420
Chromium	103	0.015	0.330
Cobalt	35	<0.0005	0.060
Copper	103	0.09	10.00
Iron	104	1.58	22.00
Lead	104	0.79	8.60
Manganese	103	0.10	9.98
Molybdenum	85	<0.005	0.78
Nickel	102	0.034	0.460
Tin	85	0.02	0.50
Titanium	104	0.04	1.10
Vanadium	99	0.050	2.200
Zinc	99	0.67	58.00
Gross beta radioactivity	323	(0.8 pCi/m³)	(12.4 pCi/m³)

[a] No individual sample analyses performed.

Certain contaminants found in total particle accumulations may be related to characteristic community activities. For example, high average ambient concentrations of vanadium are found in communities characteristically using residual fuel from certain oil fields; iron and manganese are present in significant concentrations in communities having ferromanganese blast furnaces; and annual average suspended lead particle concentrations vary directly with the annual gasoline sales from community to community. Similarly, the trend of sulfate concentration follows directly the trend of total particle concentration in communities which derive large amounts of energy from fuels with high sulfur content.[18]

2. Dustfall

Particles larger than 10 to 15 μm settle out of the atmosphere rapidly, with the settling rate dependent on particle characteristics and on atmosphere stability. Collected samples of such material are called "dustfall" or "grit and dust." Measurements usually are expressed as dust weight deposited on a unit area in a time unit (usually one month).

Typical dustfall values for urban areas are 0.35 to 3.5 mg/cm^2-mo (10 to 100 $tons/mi^2$-mo), while values approaching 70 mg/cm^2-mo (2,000 tons/mi^2-mo) have been measured near especially offensive sources. There is no question that, within a given city, dustfall tends to increase with the increase in human activity and that dustfall measurements from appropriate locations are useful for providing evidence against specific sources of dust. Attempts to use dustfall amounts as more than a crude index of air pollution is unwarranted.

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Chapter 2

EFFECTS OF ATMOSPHERIC PARTICULATE MATTER ON SOLAR RADIATION AND CLIMATE NEAR THE GROUND

A. INTRODUCTION

Particles in the atmosphere play several roles in the behavior and determination of the weather. Among the most obvious is the effect they have on radiation from the sun. They scatter the light to greater or lesser extents in different wavelength regions, depending on their size, character, and concentration, and thus provide the sky with its variable hues, its colorful dawns and sunsets, and also dense hazes and dark urban palls. More subtle changes are caused by particles through the reduction of the amount of solar energy that reaches the ground.

Particles play a less advertised but essential role in the formation of clouds. Without particles to serve as nuclei, liquid water clouds could not form except at supersaturations of several hundred percent. However, the sort of particles required (called condensation nuclei) are provided in overabundance by natural processes.

The kinds of particles which cause precipitation from clouds, in contrast to those necessary for cloud formation, are frequently in short supply in the atmosphere. In warm (above freezing) clouds, the requirement is a wide distribution of condensation nuclei, some of which are giant hygroscopic nuclei larger than 1μm. These "giants" promote the rapid growth of cloud droplets by producing some droplets large enough to fall with respect to the others. These grow rapidly by sweeping up smaller drops and very soon become massive enough to fall from the base of the cloud as rain.

Another kind of particle is required to stimulate the rapid transformation of cloud droplets into precipitation in supercooled (subfreezing) parts of clouds. Such particles are termed freezing nuclei. Without these, the water droplets would not freeze except at temperatures below -40° C. Once frozen, small ice particles grow rapidly at the expense of the surrounding water droplets and begin to fall as snow. They may later melt to become rain. Dramatic changes in cloud structure have been achieved by seeding supersaturated clouds with appropriate freezing nuclei. There is evidence, discussed below, that some observed changes in precipitation patterns in a few sections of the country can be ascribed to the inadvertent seeding of clouds by industrial contaminants.

Finally, tentative as our current information and understanding may be, the long-range potential effect of adding more and more particles to the atmosphere cannot be ignored. As more is learned about the general circulation of the atmosphere and the delicate balance between incoming and outgoing radiation (the "throttle on the atmospheric engine"), it seems increasingly possible that small changes such as those occasioned by increasing particle loads in the atmosphere may produce very long-term meteorological effects.

B. EFFECTS OF PARTICULATE MATTER IN THE ATMOSPHERE ON VISIBLE RADIATION

Nonreactive, nonhygroscopic particles of negligible fall velocity are probably the most common and persistent air pollutants. Their optical effects in producing haziness, atmospheric turbidity, and a reduction of visibility which hampers the safe operation of aircraft and motor vehicles, are well known. (See Chapter 3.) The solar radiation transmissivity ($I[\lambda]/Io[\lambda]$ where $I[\lambda]$ is the intensity of the normal-incidence solar radiation and Io[λ] is the extraterrestrial value at this wavelength) varies with height and is strongly influenced by the dust loading in the lower atmosphere.[1] Figure 2-1 illustrates the relation of transmissivity at λ = 5,000 Å to height in the atmosphere above an urban area under varying conditions of particulate loading. In the case of heavily polluted air, this radiation may be reduced by more than one-half in the lowest 300 meters of the atmosphere. There is also an increased attenuation of visible radiation near the ground in clean air, but the amount is small by comparision.

It is well known that the reduction or attenuation of visible radiation in industrialized urban areas and that the attendant gloom caused by excessive concentrations of suspended particles in the air, create a need for additional artificial illumination in offices, factories, and homes and produce added economic stresses. This is particularly true in winter when the days are shorter and the particulate content of the urban air is greater because of greater combustion of fuels. Sheppard[2] presents a comparison of the particle concentrations measured at Kew Observatory with the ratio of the visible radiation received in Central London to that received at Kew. These data are graphed in Figure 2-2. The Kew Observatory is 13 miles WSW. of Central London and the figure shows that during the winter months when the particle concentrations are high at Kew, and presumably higher in London, the visible radiation is attenuated more over Central London than over Kew. During the summer months when the particle concentrations are low at Kew, the differences in the visible radiation at the two places are slight. As another example, the average loss of sunlight in the city of Leningrad compared to the countryside was estimated to

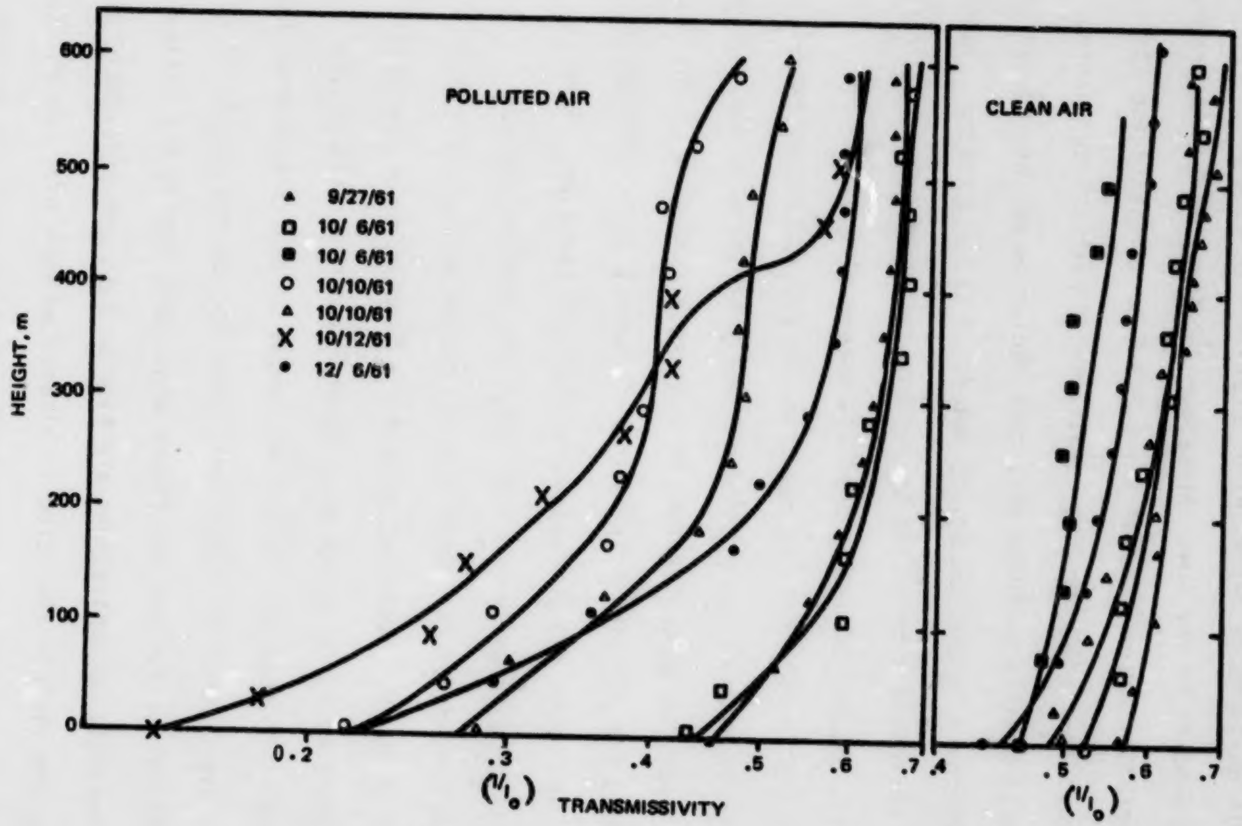

Figure. 2-1. Relation of solar transmissivity to height above ground in "polluted" and "clean" areas.[1] (This figure illustrates the effect of pollution on the attenuation of solar radiation reaching the ground.)

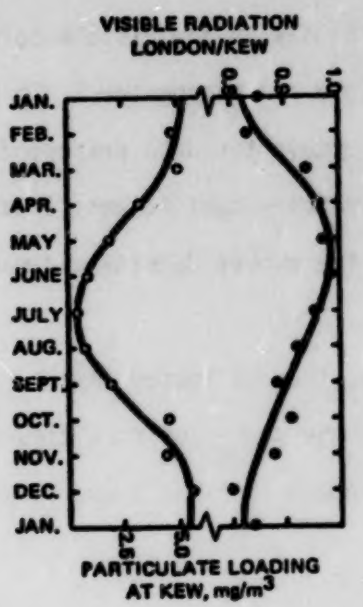

Figure 2-2. Annual variation of the ratio of visible radiation levels received at central London to that received at Kew (•), and of concentration of particulates at Kew (°).[2] (This figure indicates the relative attenuation of light at two sites in a large city.)

be 40 percent over the period of a year; in winter the loss was estimated to reach 70 percent in the city, while in summer the loss was about 10 percent.[3]

Haze in the atmosphere due to forest fires, dust storms, agricultural burning, or smoke from urban sources may become so concentrated at times that the sun appears red in the sky despite the absence of clouds. Just after sunrise or just before sunset, the haze may reduce the intensity of the direct sunlight so much that one can look directly at the sun without eye strain or injury.

Scattering of the sunlight by the particles makes the air seem "turbid." One instrument that has been used to provide a quantitative measure of the effect of turbidity in the visible portion of the solar spectrum at 5,000 Å is the Volz sun photometer.[3] This instrument was used by McCormick and Baulch[1] to obtain the data presented in Figure 2-1. These authors related the turbidity coefficients obtained at ground level and at 200 meters, to the number density and mass of particles in the intervening layer.

From scattering theory, they estimated that at any height, z, up to 200 meters above the ground, the number of particles per cubic meter, n'(z), in the radius range between 0.7 and 1 μm, is given by

$$n'(z) = 17.3 \times 10^9 B_o e^{-0.00346z} , \qquad (2\text{-}1)$$

where B_o is the turbidity coefficient at ground level as defined by Volz.[4] This relationship applies for light wind, clear sky conditions in a city during moderate-to-heavy pollution situations. In terms of mass loading near the ground, [m'(o)],

$$m'(o) = 10^3 B_o (\mu g m^{-3}) . \qquad (2\text{-}2)$$

C. EFFECTS OF PARTICULATE MATTER IN THE ATMOSPHERE ON TOTAL SOLAR RADIATION

Landsberg[5] reports that cities in general receive from 15 to 20 percent less insolation (on a horizontal surface) than do their rural environs. Insolations, as used here, means the total solar radiation received at the earth's surface per unit area per unit time. This discussion considers the part that airborne particles take in the diminution of insolation received by cities.

1. Physical Factors

The attenuation of solar radiation through the atmosphere is caused by a number of factors.[2,6-10]

a. scattering of radiation by the air molecules known as Rayleigh scattering, and by particles that are much smaller than the wavelength of the solar radiation;

b. selective absorption by the gaseous constituents of the atmosphere (e.g., ozone and CO_2) and by water vapor; and

c. scattering and absorption by atmospheric dusts and particulate matter of size greater than 0.1 μm.

The attenuation of solar radiation by water vapor and ozone is slight in the visible wavelength region usually studied.[6,7,11]

The scattering of light by aerosol particles in ambient air is a complicated process. Part of the incident light is transmitted, part is reflected in all directions either at the front surface of the particle or at an internal discontinuity, and part is absorbed. The transmission factor for scattering is a function of the wavelength of the incident light and the size and refractive index of the particle. In simple cases where a spherical form can be assumed and the size and composition of the scattering particles are known, the factor can be derived on a theoretical basis by means of the scattering theory of Gunstav Mie.

Diffuse radiation is an important factor in the amount of heat and light received at any given location.[3,10] The intensity and spectral distribution of direct sunlight and scattered daylight, and the variation of intensity with time of day, season, latitude, altitude, and atmospheric conditions (such as turbidity), are important because they affect photosynthesis in plants, the distribution of plants and animals on earth, the weathering of natural and manmade materials, the climate, and the illumination for human activity.[9] The percentage of direct solar radiation which will reach the earth's surface after its attenuation by smoke and other atmospheric constituents depends on the atmospheric turbidity, on the water vapor content of the air, and on the altitude of the sun above the horizon.

The angular pattern of Mie-type scattering is such that a large proportion of the radiation is scattered in the forward direction. Thus in the case of energy scattered from the direct solar beam, most of it will reach the earth's surface as diffuse radiation. However, in cases of large concentrations of particles--that is, when the turbidity is high--the backscatter of radiation from the direct solar beam and the absorption by the particles may be sufficiently large to markedly decrease the total solar radiation that reaches the surface.

A study by Shrader et al.[12] presents data on the relationship between the aerosol concentrations and the solar radiation that reaches the surface, as shown in Table 2-1.

Available data are sparse, but one study[12] indicates that ultraviolet intensity decreased by about 7.5 percent for each 100 $\mu g/m^3$ increases in aerosol content of the atmosphere with an average deficiency of over 20 percent in the city, compared with its environs in winter. Some studies suggest that a 5-percent reduction in total solar radiation resulting from smoke almost completely eliminates the ultraviolet component.[3,12] Even in a comparatively clean atmosphere,

Table 2-1. APPROXIMATE ASSOCIATION BETWEEN ATMOSPHERIC AEROSOL CONCENTRATIONS AND RELATIVE SOLAR RADIATION LEVELS[12]

Aerosol concentration	Solar radiation, percent of value for 100 μgm^{-3}	
μgm^{-3}	Total %	Ultraviolet %
50	105	104
100	100	100
200	[illegible]	92
400	90	77

the ultraviolet intensity drops to very low values when the sun's elevation is below 30°.[13]

The net effect of the human contribution to turbidity on surface temperature is uncertain. Mitchell[14] estimates that the effect will be a cooling trend of about 0.05° C per decade. However, this is dependent on the magnitude of the increase in atmospheric CO_2 which results from the combustion of fossil fuels and which would by itself have a warming effect.

2. Seasonal Variations

The concentration of suspended particulate matter which ranges from less than 60 to 1,700 $\mu g/m^3$ in various American cities shows notable annual variations.

Particle concentrations at ground level are greatest in winter and least in summer. However, and perhaps paradoxically, the atmospheric turbidity measured at a wavelength of 5,000 Å is greatest in summer and least in winter, according to a study by Flowers, McCormick, and Kurfis.[15] This may be due to the effect that higher summertime humidity has on the size and consequently due to the scattering efficiency of particles and also to the fact that the surface-based mixing layer, where most of the manmade aerosol is located, is deepest in summer. This later feature could explain both the lower particle concentrations observed in summer and the apparently greater total number of particles within the mixing layer that cause the higher summertime turbidities.

Lawrence[16] has shown an increase in the monthly mean maximum values of total direct solar radiation measured at Kew Observatory over two successive decades 1948-57 and 1958-67 for the months of December, January, and February. He suggests that this increase presumably can be explained by the reduction of smoke resulting from the effectiveness of the Clean Air Act of 1956. Mean values of total direct solar radiation for all other months and for the year showed a decrease for the second decade, which cannot be similarly explained.

Landsberg[5] has summarized the radiation loss data compiled by Steinhauser et al.[13] for three Central European cities--Frankfurt, Leipzig, Vienna--and their adjacent rural areas, for the four seasons of the year. There are contrasts in loss of solar radiation between spring or summer and winter, as will be seen in Table 2-2. Steinhauser and coworkers[13] also reported that Vienna receives less total radiation than the nearby countryside: in winter, 85 percent of the total solar radiation of suburban Hohe Warte; in spring, 92 percent; in summer, 92 percent; and in autumn, 87 percent.[8,13]

3. Weekly Variations

In addition to the seasonal and diurnal variations or patterns of total solar radiation in urban communities, a weekly cycle of intensity of total solar radiation has been observed,[17-19] which is related to

Table 2-2. LOSS OF SUN'S RADIATION IN THREE EUROPEAN CITIES OVER THAT IN THE ADJACENT COUNTRY[5]

Season	Solar Elevation			
	10°	20°	30°	45°
	Radiation Loss, %			
Winter	36	26	21	--
Spring	29	20	15	11
Summer	29	21	18	14
Autumn	34	23	19	16

the weekly cycle of industrial and commercial activity.

Mateer,[19] in a study of the total solar radiation at Toronto during 1937 to 1960, found that the average radiation received on a weekday was about 2.8 percent less than that received on Sunday. The probability of obtaining such a difference in radiation by chance was less than 0.5 percent. Thus, while a real difference in radiation exists between Sundays and weekdays, the magnitude is rather small.

D. INFLUENCE ON PRECIPITATION

There is evidence that some of the particles introduced into the atmosphere by man's activities can act as nuclei in processes which affect the formation of clouds and precipitation.

Condensation nuclei in the size range greater than 1μm are often of hygroscopic particles.[20,21] Because of their affinity for water, these particles play an important role in the transformation and condensation of water vapor into liquid water droplets or solid ice particles, and are of vital importance in the formation of fog, clouds, and rain. Combustion products of man's industry and technology, as well as volcanic eruptions and ocean spray, are significant sources of these nuclei. The excess production of condensation nuclei in the air over cities is a long-established fact,[5,21,22] and has been reaffirmed with many amplifying circumstances.[22,23] Schaefer's[24] discussion of the abundance of nuclei downwind of industrial areas and areas of agricultural burning and forest fires, points out that an overabundance of nuclei tends to inhibit rather than promote precipitation. Evidence has been presented that giant nuclei, which may initiate the coalescence process, are more abundant in industrial areas than elsewhere.[18]

The diurnal, weekly, and yearly cycles of both suspended particle concentrations and dustfall correspond closely to man's pattern of activities and combustion requirements. There are usually two diurnal peaks (see Figure 2-3[17]), greater midweek concentrations compared to Sundays, and greater mean concentrations in winter than in summer. (See Figure 2-4.[18])

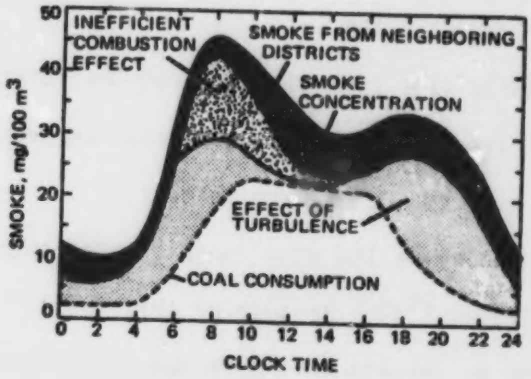

Figure 2-3. Causes of cyclical diurnal smoke variations.[17] (This figure shows causes of changes in the diurnal airborne particulate concentration in an urban location.)

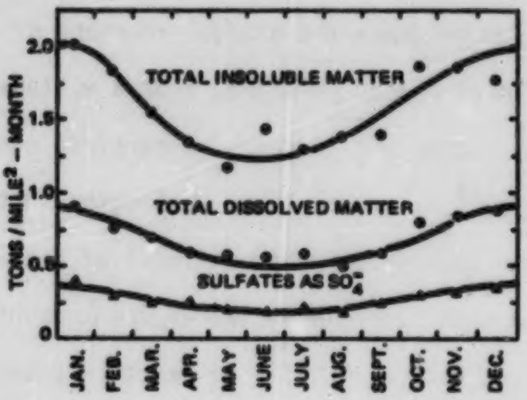

Figure 2-4. Yearly cycle (averaged over 6 years) of deposited pollutants.[18] (This figure illustrates the variation of amount of pollutants deposited at Leicester townhall averaged over the 6 years ending March 1939 and shows the greater mean concentration in winter as compared with summer.)

A correlation has been found between patterns of precipitation over cities and the adjacent countryside and the variations of particle concentrations in the atmosphere.[22,23,25] The influence of cities on precipitation is complex; however, there is a general tendency for urban factors to increase precipitation.[22] These factors, not necessarily in order of importance, are:

1. water vapor addition from combustion sources and processes,
2. thermal updrafts from local heating,
3. updrafts from increased friction turbulence,
4. added condensation nuclei leading to more ready cloud formation, and
5. added nuclei which may act as freezing nuclei for supercooled cloud particles.

Landsberg[22] cites as evidence the gradual increase of rainfall which followed the growth of Tulsa, Oklahoma, from a village to a city in five decades and the concomitant increase in particle concentrations. A study by Kline and Brier[26] in metropolitan Washington, D.C., indicates that there is a considerably higher level of freezing nuclei in the metropolitan area than there is in the adjacent countryside. Schaefer[24] has pointed out the possibility of reaction between lead particles in the atmosphere from combustion of leaded gasoline and iodine from the burning of wood and other organics to form lead iodine nuclei for ice formation.

Ashworth[25] first suggested the correlation between the weekly cycle of smoke in industrial areas and that of precipitation. Frederick,[27] in a more recent analysis, showed a definite minimum Sunday rainfall for a 10-year period in Louisville, Pittsburgh, and Buffalo.[22,27] Precipitation occurred in these less often on Sundays than on other days of the week, and the average rainfall was less for Sundays than for weekdays. A strong city influence is also suggested in the snow patterns in Toronto,[28] and Atkinson[29] has shown the influences of London on the precipitation from a thunderstorm. This latter analysis attributed the influence to increased moisture and heat rather than to particles.

An interesting and significant increase in precipitation has been observed at La Porte, Indiana, since 1925. La Porte is 48 km (30 statute miles) east of the large complex of heavy industries in the metropolitan Chicago area. Changnon[30] compared precipitation at La Porte, Valparaiso, and South Bend, Indiana, with a 5-year moving average of the number of smoky and hazy days in Chicago (Figure 2-5). The temporal distribution of the smoke-haze days after 1930 is rather similar to the La Porte curve. A notable increase in smoke haze days began in 1935, becoming more marked after 1940, coincident with the sharp increase in the La Porte precipitation curve. The reduction in the frequency of smoke-haze days after the peak reached in 1947 also generally matches the decline of the La Porte curve since 1947.[30]

Stout[31] has shown that the shape of the time-series curve for La Porte precipitation also generally matched a time-series curve for annual steel production in the Chicago industrial complex. Between 1905 and 1965, peaks in steel production, which occurred when production in most other industries was also high, were all associated with high points in the La Porte precipitation curve.

The existence of a precipitation anomaly at La Porte has been challenged on the basis of some possible procedural errors in the measurements,[32] but it has been supported through analysis of stream flow data.[33]

The effect of industrial pollution on precipitation has also been studied by Telford.[34] He found that smoke from steel furnaces was a prolific source of freezing nuclei, increasing counts by a factor of 50 over those in nearby clean air. He concluded that there should be increased rainfall downwind of such installations.

While the foregoing relationships between urban pollution sources and clouds and precipitation are valid, Twomey[35] has recently presented results of studies which suggest that neither particulate pollution nor the classically proposed sea salt particles form the major source of

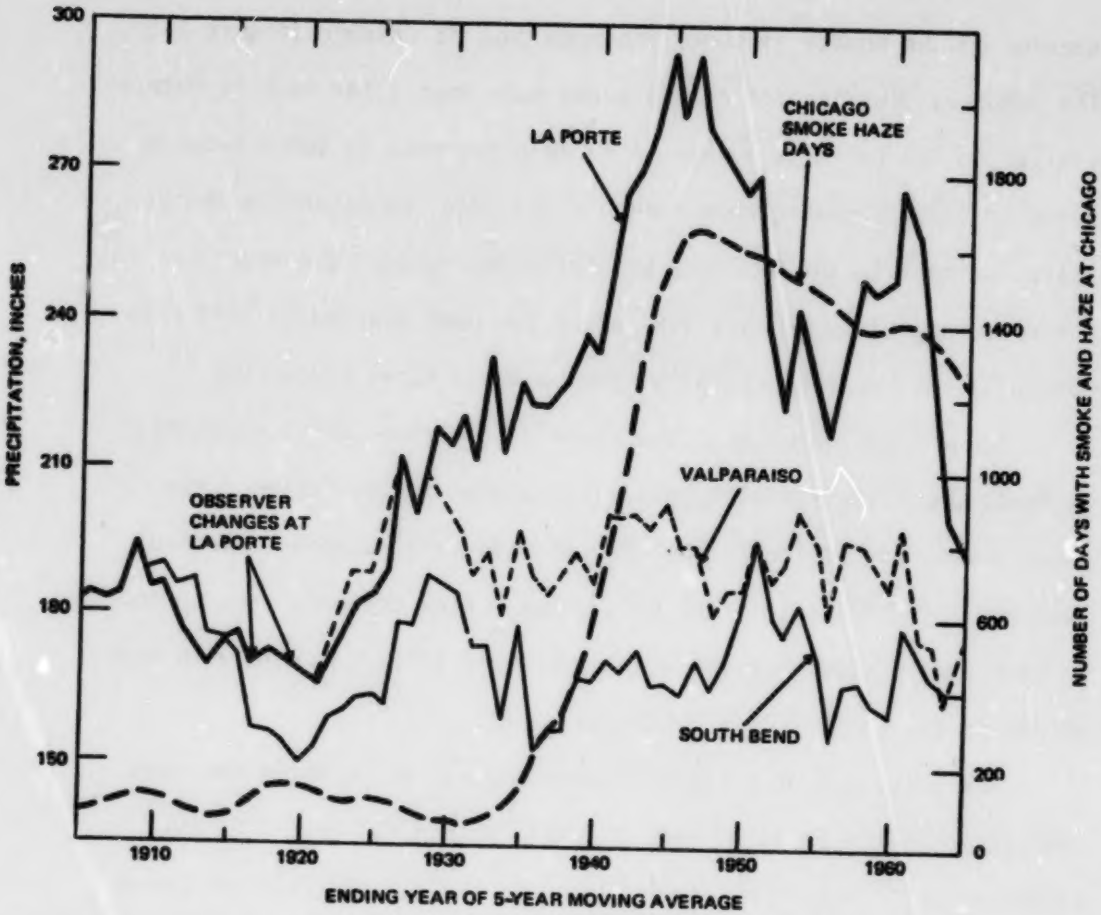

Figure 2-5. Precipitation values at selected Indiana stations and smoke-haze days at Chicago.[30] (This figure shows the way in which precipitation trends at La Porte follow the haze changes in Chicago. The results are plotted as 5-year moving averages.)

cloud droplet nuclei. Rather, he suggests that ammonium sulfate particles produced in the atmosphere from ammonia and gaseous sulfur compounds are the most prevalent nuclei. The similarity in number of these nuclei found in the northeastern United States and southeastern Australia discounts the possibility that industrial pollution plays a large part in the formation of these particles.

E. RELATION TO WORLDWIDE CLIMATIC CHANGE

Theoretical considerations and empirical evidence indicate that atmospheric turbidity is an important factor in the heat balance of the earth-atmosphere system. The observed increase in turbidity over the past few decades may play a role in the reported decrease in worldwide air temperature since 1940 by increasing the planetary albedo.[7,36]

Angstrom[37] estimated roughly that a change in the albedo from 0.40 to 0.41 corresponds to a change in the mean temperature of the earth-atmospheric system of close to 1° C. Humphreys[38] made similar calculations with roughly the same results, and also showed that the interception of outgoing radiation by fine atmospheric dusts is wholly negligible in comparison with the interception of incoming solar radiation. Temporal and spatial changes in the atmospheric turbidity of 100 percent, corresponding to albedo changes of 10 to 15 percent, from one day to the next or from one locality to another, are very commonplace.[36] Even though these figures may well overestimate the actual changes brought about in atmospheric temperatures, the course of atmospheric turbidity over the earth is an important climatic factor.

There are data available[36,39] from which the trend in turbidity during the past century can be estimated. Angstrom gave 0.098 as the value of the mean annual turbidity at Washington, D.C. (1903 to 1907),[11] and 0.024 as the value at the Davos Observatory, Switzerland (1914 to 1926). The values for Washington were determined from data on solar transmission (by wavelength) published by the Smithsonian Institution; those for Davos were from data attributed to Lindholm on dust absorption. In 1962, determinations of the atmospheric turbidity were begun at the Continuous Air Monitoring Program station[36,40] of the Public Health Service, near the Smithsonian Institution; the mean annual turbidity recorded for 1962 to 1966 was 0.154,[40] a 57-percent increase over the 1903-to-1907 value. From 1957 to 1959, determinations of atmospheric turbidity were again made for Davos by Valko[41] and were given as 0.043, a 70-percent increase over the 1914-to-1926 value.

When the scattering theory with a Junge distribution of particle size[4] is used, the values of turbidity change imply an increase in the average annual number of aerosol particles in the range of 0.1 to 1 μm radius, of $2.8 \times 10^7/cm^2$ and $0.95 \times 10^7/cm^2$ over Washington and Davos respectively, during the periods shown. Nearly two-thirds of the Washington increase might be attributed to the increased population and urbanization of the District since the turn of the century. A significant remainder, however, as judged by the Davos increase, may be indicative of a much more general buildup of atmospheric aerosol.

When the above facts are put together, they indicate that for Washington, D.C., during the period 1903 to 1966, there has been a possible decrease of nearly 3 percent in the total available solar energy at ground level and a possible increase in the average annual number of aerosol particles of $2.8 \times 10^7/cm^2$. The net effect of this apparent secular increase in turbidity (which from the Davos, Switzerland, and other evidence[39] appears likely to be worldwide) is probably to increase the mean albedo of the planet and to reduce the mean temperature of the earth-atmosphere system.

The increase in atmospheric turbidity consequent upon volcanic eruptions may have temporary effects on atmospheric temperatures. Mitchell[42] concluded that temperatures over large areas of the world may be depressed by 0.5° F or more in the first or second year following an unusually violent eruption. However, McCormick and Ludwig[36] suggest that the effects of man's pollution of his environment are increasing steadily along with the world population. The emission of long-lived particles, keeping pace with the accelerated worldwide production of CO_2, may well be leading to the decrease in world air temperature in spite of the apparent buildup of CO_2.[43]

Mitchell[14] presents a comprehensive though necessarily qualitative review of factors affecting the global temperature and concludes that at the present rates of injection of CO_2 from fossil fuel combustion and of human-derived particles, a cross-over of effects (CO_2-warming; particle-cooling) will not obtain until after 2,000 A.D.

Charlson and Pilat[44] have pointed out that, although absorption of energy by airborne particles is usually neglected in comparison with the effects of scattering on the attenuation of solar energy reaching the earth's surface, heat gained by particles may be significant in determining the heat balance of the atmosphere. Further data are needed on the relative magnitudes of scattering and absorption coefficients of both industrial pollutants and naturally occurring materials such as volcanic dusts.

F. SUMMARY

Atmospheric particles scatter and absorb light from the sun, thus reducing the visible radiation available to cities and the solar radiation that reaches the earth. The average year-round illumination (i.e., the portion of the spectrum that is visible to the eye) may be reduced by one-third or more in some cities. Daylight illumination in center of London, for example, was found to be 20 percent less than that found at a slightly less polluted part of the city quite near the center.

Part of the sunlight reaching the earth comes from the direct beam and part from that scattered from the rest of the sky. The total solar energy reaching the earth is the sum of both the direct and the scattered radiation. In general, cities receive 15 to 20 percent less total solar radiation than do rural environments, although the reduction may be considerably greater under some circumstances. For a typical urban area in the United States, with a geometric mean annual concentration of roughly 100 $\mu g/m^3$, the total sunlight is reduced approximately 5 percent for every doubling of particle concentration. This effect is more pronounced on the ultraviolet portions of the spectrum.

Diurnal, weekly, and yearly cycles of both suspended particles and dustfall correspond closely to man's pattern of activities. There are usually two daily peaks, greater midweek concentrations compared with Sundays, and greater mean concentrations in autumn and winter than in spring and summer. The seasonal variations are due largely to the use of coal and heavy fuel oil for heating purposes. Solar radiation attenuation patterns also show weekly and seasonal variations. On weekdays, the attenuation is slightly more than on Sundays, and the losses in intensity of the direct beam during its passage through an atmosphere polluted by smoke may become as high as one-third in the summer and two-thirds in the winter. Reductions in the intensity of solar radiation or changes in its spectral distribution have significance for the photosynthesis of vegetation, the distribution of plants and animals on the earth, the weathering of natural and manmade materials, and man's aesthetic enjoyment and physical well-being.

The increased emission of fine particles into the atmosphere also may cause changes in the delicate heat balance of the earth-atmosphere system, thus altering worldwide climatic conditions. The rise in atmospheric turbidity increases the planetary reflectivity, or albedo, and reduces the solar energy available to maintain surface temperatures. This phenomenon may be responsible for the reported decrease in worldwide temperature since the 1940's. Comparisons at different sites over the world covering periods as long as 50 years suggest that a general worldwide rise in turbidity may be taking place. The increasing levels of atmospheric carbon dioxide resulting from man's combustion of fuels probably exerts an opposite effect on worldwide temperatures. It has been speculated that the emission of long-lived particles to the air may gradually depress world air temperature despite the apparent buildup of carbon dioxide.

Some of the particles introduced into the atmosphere by man's activities also can affect the weather by serving as condensation nuclei that influence the formation of clouds, rain, and snow. The large airborne particles generated in metropolitan areas serve as a base for the condensation of moisture and lead to the rapid formation of rain droplets or ice crystals. Patterns of precipitation over cities and the adjacent countryside have shown a correlation with presumed particle concentrations in the atmosphere. Some cities display a definite minimum rainfall on Sundays, when particle concentrations are usually lowest, and records over several decades reveal that rainfall levels may increase with the concomitant rise in particle concentration that generally accompanies urban growth. In addition, long-term changes in the frequency of smoke-haze days in one city may be reflected in rainfall levels in a nearby downwind city.

Thus airborne particles can, through a number of mechanisms, influence man's surroundings and have considerable impact on weather and climatic conditions.

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Chapter 3

EFFECTS OF ATMOSPHERIC PARTICULATE MATTER ON VISIBILITY

A. INTRODUCTION

One of the dramatic effects of air pollution is a degradation of the visibility. Visibility in the atmosphere is reduced by two optical effects which air molecules and aerosol particles have upon visible radiation. One is the attenuation by the molecules and particles of the light passing from object to observer. It is the result both of absorption of light and of the scattering of light out of the incident beam. The light received from the object and that received from its background are diminished by this attenuation, and the difference between the two (contrast) is consequently diminished with the result that the eye's ability to distinguish the object from its background is reduced. The other optical effect that degrades the contrast between object and background is the illumination of the intervening air which results when sunlight is scattered into the line of sight by the molecules and particles in the line of sight. It is a common observation that dark objects become progressively lighter in shade as they become more distant. The most distant mountain that one can distinguish, typically, is almost as light or bright as its sky background.

In those cases where a clear-cut relationship can be shown to exist between visibility and the mass of suspended particles per volume of air (mass concentration), it is possible to use some of the meteorological visibility records to infer the amount of pollution in past years as well as to study trends. In fact, many such studies have been made even without a substantive knowledge of the relationship. Holzworth[1] used existing visibility data both as an indication of the amount of pollution for comparison of an urban area (Columbus, Ohio) with a rural area, and for the qualitative inference of trends of both increasing and decreasing amounts of smoke or other atmospheric aerosol. Robinson[2] also discussed the use of meteorological visibility records and their interpretation. Both Holzworth[1] and Robinson[2] demonstrated that visibility degradation can be associated with air pollution. Neither, however, developed the sort of correlation between mass of suspended particulate matter per volume of air and visibility that is needed for air quality criteria. Robinson[2] indicated doubt in the existence of a generally applicable relationship between mass concentration and visibility. This doubt was based on experimental results as well as on the complexity of the problem. Recent experimental and theoretical advances make possible some useful conclusions, and this chapter will define a simple relationship between mass of suspended particles and visibility, specify the circumstances under which it can be expected to be reliable, and describe the conditions which are too complex for this simple treatment.

The words visibility and visual range are usually used synonymously to mean the distance at which it is just possible to perceive an object against the horizon sky. Middleton[3] reports that the originator of both terms intended that only visual range be considered as a distance, while visibility should convey a more qualitative judgment about the clearness of seeing. In this chapter, however, the terms visibility and visual range will be used interchangeably to mean distance.

B. IMPORTANCE OF ATMOSPHERIC AEROSOLS TO THE GENERAL OPTICAL PROBLEM

Decreased visibility obviously interferes with certain important human activities, such as the safe operation of aircraft and automobiles and the enjoyment of scenic vistas. The effect of decreased visibility on the large-scale operations of commercial aircraft in metropolitan areas is a problem of growing concern. Regulations prescribing limitations on aircraft operation become increasingly severe as visibility decreases below 8 km (5 statute miles).[4] Most restrictions are invoked when the visibility is 5 km (3 statute miles) or below. In areas with a high density of aircraft traffic, visibilities much below 8 km tend to slow down operations by requiring larger separations between aircraft. Even though most commercial airplanes always fly under Instrument Flight Rules rather than Visual Flight Rules, good visibility increases both the safety and permitted traffic density. With ground visibilities less than 5 km, airports in the United States are closed to airplanes (or pilots) without instrument flight capabilities.

A 1963 report by the Civil Aeronautics Board to the Committee on Public Works, U.S. Senate,[5] states that records of both automobile and aircraft accidents show cases where poor visibility due to smoke and air pollution was an important causal factor.

C. PHYSICAL RELATIONSHIPS BETWEEN VISIBILITY AND PARTICLE CONCENTRATION

Many derivations of visibility theory have been published. Although Robinson's[2] approach is directed towards the air pollution problem, Middleton's[3] book presents a more complete view of the problem of atmospheric clarity.

For the simplest case of attenuation of a light beam along its path, the intensity, I, decreases by an increment, dI, over the increment of path, dx. The relationship between intensity and distance is:

$$\frac{dI}{I} = - bdx \qquad (3\text{-}1)$$

or, in integrated form,

$$I = I_0 e^{-bx} \qquad (3\text{-}2)$$

where b is the extinction coefficient assumed to be constant over x, and I_0 represents the intensity of light at x = 0.

The extinction coefficient, b, is the sum of four terms:

1. the scattering coefficient of the air molecules, $b_{Rayleigh}$;
2. the scattering coefficient of particles or aerosol, b_{scat};
3. the light absorption by gases, $b_{abs-gas}$, and
4. the light absorption by the aerosol, $b_{abs-aerosol}$.

Of these four, the scattering due to aerosol is usually assumed to dominate in haze.[2] The process of scattering amounts to the removal of light from the original beam and its redistribution in different directions. Scattering thus differs from absorption in which the light energy is lost to the absorber; in scattering, the light energy is only spatially redistributed. Nonetheless, this removal of light from the beam (or line of viewing) does result in extinction. For the purposes of this discussion, visual range is defined for an "average" human eye and a 2-percent contrast threshold, by the following equation:[2,3]

$$L_v = \frac{3.9}{b_{scat}} . \qquad (3\text{-}3)$$

Here, L_v is the visual range in meters and b is, as before, the extinction coefficient per meter along the path of sight for the case of a black object. If b is determined at a point without knowledge of the entire sight path, then L_v is "equivalent visual range"--the distance one could see if the extinction coefficient were constant along the sight path.

"Visual quality" as perceived by even the well-trained observer is not easily described. Among the complications is the fact that particles responsible for haze scatter more light in a direction close to that of the original beam (so-called forward scatter) and less light in a backward direction. Thus, the same haze may appear to be much more dense when looking toward the sun and less dense when looking away from the sun.

Besides this directional factor, there is also a wavelength (or color) dependence. Angstrom[6], Junge[7], and others have shown that the extinction coefficients of haze in general are inversely proportional to a power of wavelength:

$$b = \frac{1}{\lambda^{\alpha}} \tag{3-4}$$

where α has measured values of around 1.0 to 1.5. This relation indicates that blue light (of shorter wavelengths) will be scattered to a greater degree than red light (of longer wavelengths). It is for this reason that the sun's disc, when observed through a haze that is dense enough to permit such viewing, appears red, orange, or even brown even though light absorption is not necessarily occurring.

D. COMPLICATIONS AND LIMITATIONS IN THE VISIBILITY PROBLEM

1. Natural Aerosols and Hazes

The general problem of natural particulate matter--from whatever source--must be considered. The oceans produce salt particles, trees produce terpenes that may result in organic particles,[8] forest fires make smoke, and so on. Man has little hope of controlling the quality of air that enters the urban areas from uninhabited lands. Nonetheless, these aerosols sometimes cause dramatic reduction in the visual range. In order to properly evaluate the importance of natural aerosols, the visual range should be determined before the air mass enters a populated area.

2. Fogs

When the relative humidity exceeds approximately 70 percent, many types of particles exhibit deliquescent behavior and grow into fog droplets. Natural particles such as sodium chloride from the sea as well as many products of human activity can thus act as condensation nuclei (Chapter 2). The property of deliquescence and the relative humidity at which rapid and large change in particle size occurs are both very dependent on the chemical composition and original size of the particles. As a result, unless the chemical composition as a function of particle size is known for the aerosol, very little can be said about the relationship between visibility in even "thin" fog and the amount of material present as pollutant.[9]

Since in many parts of the world (particularly in moderate latitudes) the relative humidity is often above 70 percent, the effect of humidity is often considered from a practical point of view. With relative humidity as the parameter controlling the growth of particles, the expected visibility (at least up to humidities of 96%) can be estimated from particle concentrations measured below 70 percent.

E. THE LOW-HUMIDITY, WELL-AGED HAZE

1. Size Distribution

Because little deliquescence occurs below 70 percent relative humidity, the relationships to be presented here will be limited to the range of humidity from 0 to 70 percent.

Recent advances in both theory and technology have resulted in a simplification of the description of well-aged aerosols. Junge,[7] Friedlander and Wang,[10] Whitby and Clark,[11] and Peterson and Paulus[12] have shown that aerosols in the lowest region of the atmosphere (troposphere), whether over urban areas or not, tend to have similar size distributions. Figure 3-1 shows several typical size distributions to illustrate this feature.[13]

If it is assumed that this recurring size distribution exists in general, then it is possible to relate the optical properties of the haze to the amount of mass concentration of material present. This generalization may not apply to freshly formed smoke. Brief guidelines

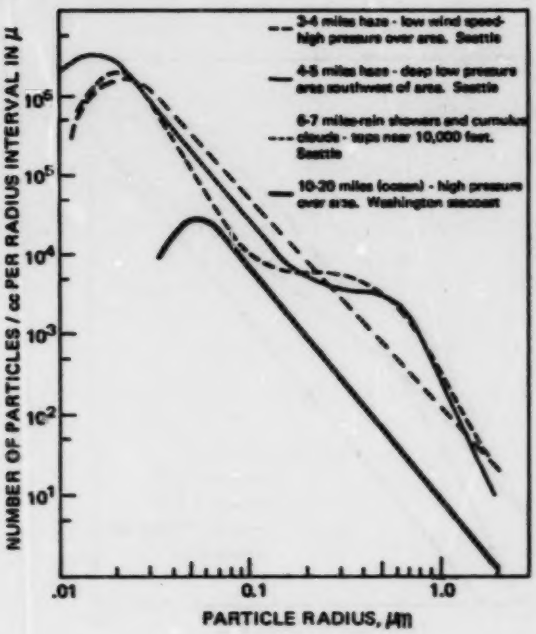

Figure 3-1. Four typical measured size distributions of atmospheric suspended particles together with the corresponding visibilities.[13] [The figure shows that aerosols in the lowest region of the atmosphere (the troposphere) tend to have similar size distributions.]

for determining when the size distribution has become sufficiently well-defined will be given later.

2. Mie Solutions

As mentioned earlier, another important assumption which is usually made is that, of the four extinction components, light scattering by aerosols dominates. Current research indicates that this is probably a justifiable assumption.[14] Figure 3-2 shows typical scattering cross sections for green light as a function of particles size for aerosol particles important in haze,[14] computed via the theory of Gustav Mie.[15] For some particle sizes, differences in the scattering coefficient of a factor of 3 exist between the two refractive indices which span the realistic range for the atmospheric case. However, Pueschel and

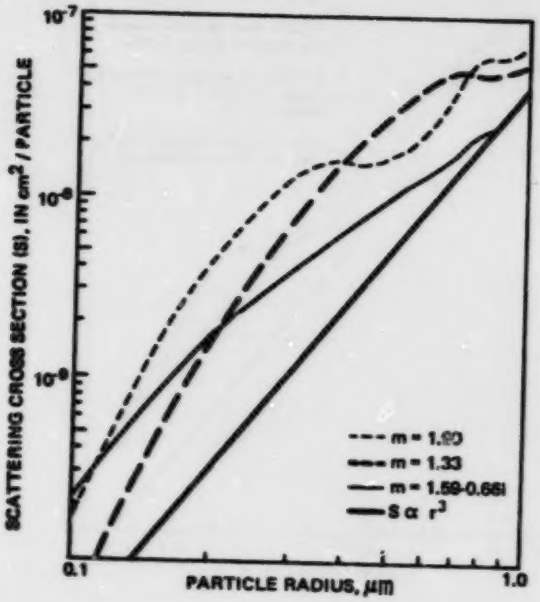

Figure 3-2. The relation of scattering cross section to particle size for particles of three different refractive indices (m).[14] (The figure shows that the scattering cross section of atmospheric particles varies roughly as the cube of their radius within the range 0.1 μm; the range of refractive indices, 1.33 to 1.60, for which the proportionality holds includes most materials found in atmospheric aerosols.)

Noll[13] conclude that the extinction coefficient of an aerosol in the troposphere is nearly independent of the refractive index of the particles if the size distribution is close to those described in Figure 3-1.

3. Dependence of Extinction on Particle Size for the Atmospheric Case

If the data in Figures 3-1 and 3-2 are broken down into narrow radius intervals (e.g., 0.01 μm), and a calculation is performed to yield the extinction coefficient for each radius interval, the particle size dependence of atmospheric extinction of green light is revealed.

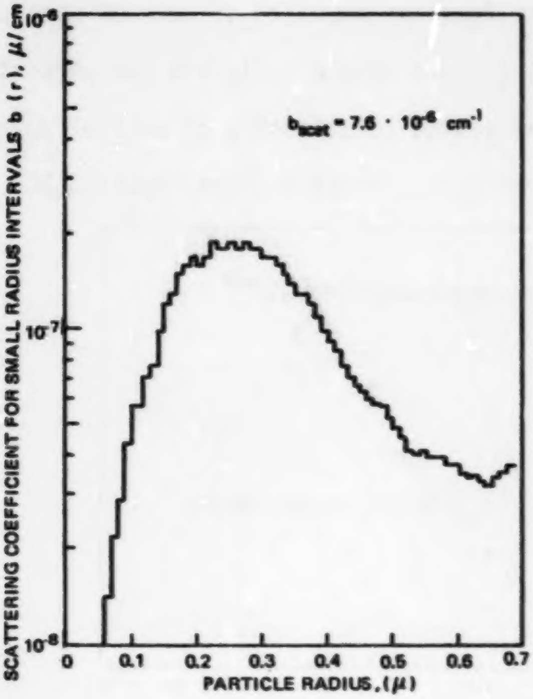

Figure 3-3. Scattering coefficients for a typical atmospheric size distribution.[13] (The figure shows the particle size dependence of atmospheric extinction obtained by breaking down the data of Figures 3-1 and 3-2, for the 5-to-6-km-haze case, into 0.01-μm intervals to yield the scattering coefficient of each radius interval.)

Figure 3-3 shows the results for a typical size distribution of spherical particles having a refractive index of 1.5.[13]

In general, this procedure shows that a narrow range of particle sizes, usually from 0.1 to 1 μm radius, controls the extinction coefficient and therefore the visibility. If the values of the extinction coefficient for each radius interval are then summed, the total extinction coefficient due to scatter (b_{scat}) is obtained.

4. The Mass-Light Scattering Relationship

It is also possible to calculate the volume of particulate matter per unit volume of air (i.e., in cubic micrometers of aerosol per cubic centimeter of air). The familiar quantity of aerosol mass concentration (μg/m^3) is proportional to this volume ratio via the particle density. Figure 3-4 shows the calculated relationship of aerosol volume (μm^3/cm^3) to scattering coefficient per meter [for green light (5,500 Å) and for a

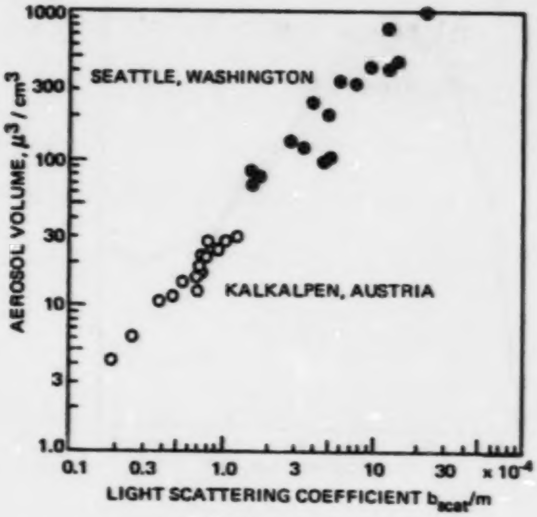

Figure 3-4. The dependence of scattering coefficient (m^{-1}) on volume of aerosol particles (μm^3/cm^3)calculated from measured size distributions.[14]

refractive index of 1.6] based on 32 individually measured size distributions and particle counts.[14] Sixteen of these size distributions used in Figure 3-4 were measured in Seattle under varying meteorological conditions. The remaining 16 were obtained in the Austrian Alps under conditions where, presumably, only natural aerosol was present. The implication of these calculations is that the volume, and thus the mass concentration of well-aged aerosol, is approximately proportional to the light-scattering coefficient for atmospheric aerosols originating naturally or as the result of man's activity.

It follows, therefore, that even though the aerosol mass is distributed over perhaps two or three decades of size and light scatter is caused by particles of one narrow size range, a proportionality can exist between the number of particles scattering light and the total mass concentration of particles. Constant shape of the size distribution here implies that for all radius intervals, the number of particles in an interval is proportional to the number in the corresponding interval in a reference size distribution.

Figure 3-5 shows data obtained by an integrating nephelometer for a wavelength of 5,000 Å confirming this calculated dependence.[14] The relationship can be summarized as follows:

$$\text{mass } (\mu g/m^3) \simeq 3 \times 10^5 \, b_{scat}/m. \qquad (3\text{-}5)$$

Figure 3-6 shows data like those of Figure 3-5 plotted in a different fashion to illustrate their distribution.[16] From equations 3-3 and 3-5, the product of equivalent visual range and mass concentration (L_v X conc) can be obtained. Since

$$L_v = \frac{3.9}{b_{scat}} , \qquad (3\text{-}3)$$

multiplying both sides of the equation by concentration gives

$$L_v \text{ X conc} = \frac{3.9 \text{ X conc}}{b_{scat}} . \qquad (3\text{-}3a)$$

The units on both sides of this equation are mass per area (e.g., $\mu g/m^2$). If we use the particular proportionality in equation 3-5, then

$$L_v \text{ X conc} \approx 1.2 \text{ X } 10^6 \ \mu g/m^2 = 1.2 \ g/m^2 . \qquad (3\text{-}6)$$

This product has a simple physical significance: it is the mass of material in a column of length L_v and one square meter in cross section. In other words, it is the amount of material per square meter between

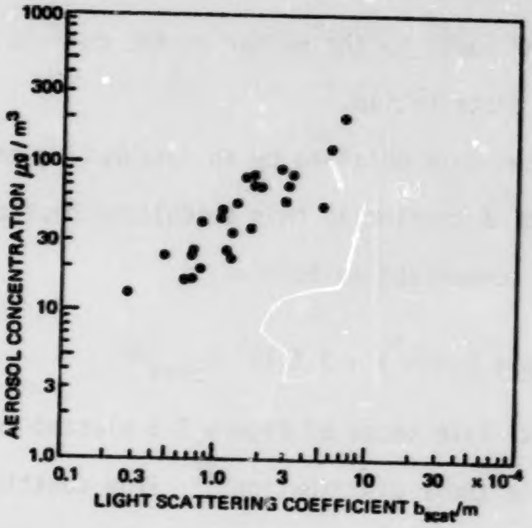

Figure 3-5. Measured dependence of mass of aerosol particles per volume ($\mu g/m^3$) on the light scattering coefficient (m^{-1}) in Seattle, November-December 1966.[14]

the observer and a point at the limit of visibility. Each point of Figure 3-5 can be used to form this number since each pair of values of mass concentration and scattering coefficient can be used to obtain a different proportionality of the sort shown in equation 3-5. Figure 3-6 consists of histograms showing the number of occurrences for different values of the quantity L_v X conc, found at various locations where both b_{scat} and mass concentration were measured. Here, since the modal value is about 1.2 g/m^2, one can write:

$$L_v \text{ X conc} \approx 1.2^{+1.2}_{-0.6} \text{ g/m}^2 \qquad (3\text{-}7)$$

to include virtually all cases. Equation 3-5 can also be rewritten to include these upper and lower limits:

$$\text{mass } (\mu\text{g/m}^3) \approx 3^{+3.0}_{-1.5} \text{ X } 10^5 \; b_{scat}/\text{m}. \qquad (3\text{-}8)$$

Noll, Mueller, and Imada[17] have presented a summary of studies relating visual range to aerosol mass concentration. Measured mass concentrations were compared with weather service observations of "visibility," soiling index (coh) values, with Voltz sun photometer readings, and with light-scattering coefficients. The study results are summarized in Table 3-1 for three levels of mass concentration.

This research area is an active one, and new data are constantly being acquired. Improvements in the understanding of these phenomena and the accuracy of descriptions will no doubt occur in the future. However, these values are known to be accurate to well within one significant figure for the cases measured, and their utility for criterion purposes is therefore clear.

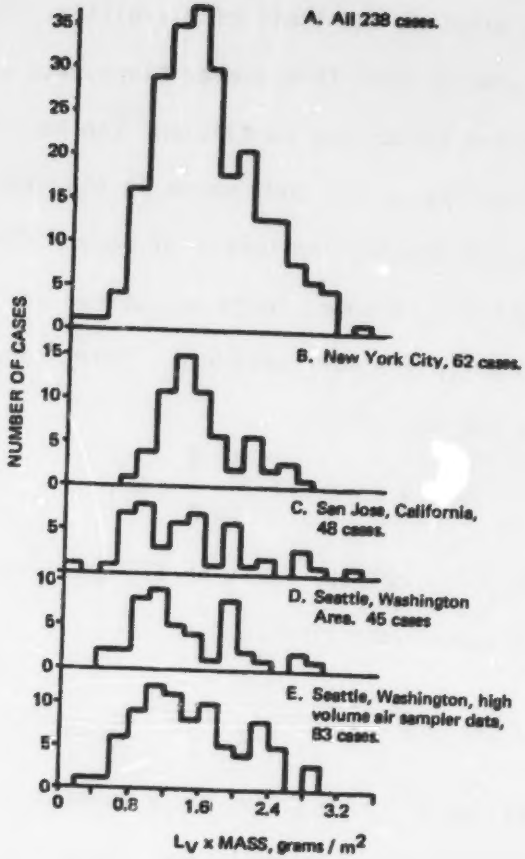

Figure 3-6. Histograms of equivalent visual range-mass concentration product at several locations.[16] (The figure shows the number of cases with a given range of values of the product of mass and equivalent visual range for data at various locations studied; this product represents the mass required to determine the visibility in a column 1 m^2 in cross section along the light path.)

Photochemically produced aerosols, as well as other organic aerosols, can be expected to have similar visibility effects if their size distribution remains reasonably constant. Since the physical processes governing size distribution are assumed to be largely independent of the chemical nature of the particles,[10] the size distribution should be similar to those shown in Figure 3-1. However, if such particles are volatile or metastable to oxidation as suggested by Goetz et al.,[18] the mass determination may be difficult. Although visibility degradation is evident in photochemical smog, experimentally determined size distribution data are needed.

It can be seen from Figure 3-5 that the practical light-scattering coefficient for 5,000 Å wavelength is 10^{-4}/m. The value 1×10^{-4}/m represents fairly clean air with a particle mass concentration of about 30 $\mu g/m^3$, while 10×10^{-4}/m represents more polluted air (concentration about 300 $\mu g/m^3$). Of course, the reciprocal relationship (equation 3-3) could be used in combination with equation 3-5.

$$L_v \text{ (km)} = 1.2^{2.4}_{0.6} \times 10^3/\text{conc } (\mu g/m^3). \qquad (3\text{-}9)$$

Table 3-1. COMPARISON OF VISIBILITY AND AEROSOL CONCENTRATION FOR FOUR STUDIES[17]

Study	100 $\mu g\ m^{-3}$		200 $\mu g\ m^{-3}$		300 $\mu g\ m^{-3}$	
	Visibility (km)	Ratio to study 1	Visibility (km)	Ratio to study 1	Visibility (km)	Ratio to study 1
1. Berkeley-Oakland	19.8	1.0	9.1	1.0	5.6	1.0
2. Volz sun photometer	17.4	0.88	7.7	0.86	4.8	0.86
3. Soiling index	10.0	0.50	4.8	0.54	3.2	0.57
4. Continuous recording instrument	12.1	0.61	6.1	0.68	4.0	0.72

F. VISIBILITY RESTRICTION WITH HIGH HUMIDITY AND AEROSOLS

As mentioned above, aerosols increase in size with increasing relative humidity; the relationship between the size change and the humidity varies with the chemical composition of the particle. This relationship can be quantified for specific aerosols of known composition and, statistically, for the mixed aerosol usually found in the atmosphere.

In the mean, the change of size of an aerosol follows the equation

$$r/r_0 = (1 - f)^{-e(f)} \tag{3-10}$$

where r_0 is the particle radius in dry air and e(f) is a value between 0.21 and 0.33.[19] For mean values of atmospheric aerosols, this equation describes, with $e = 1/4$, the growth within a humidity range of 30 and 95 percent, reasonably well.

The relative change of visibility L with the humidity f at a given constant aerosol content for the humidity range of $80\% \leq f \leq 95\%$, in the mean, may be estimated according to the equation[20,21]

$$dL/L = - \frac{df}{2(1-f)} \,. \tag{3-11}$$

The extent of the variations to be expected is made clear by the empirically established relationship between aerosol content, visibility, and relative humidity (1964 in Berlin-Germany; see Figure 3-7).[22] At constant aerosol concentration, the increase of the relative humidity from 70 to 95 percent shows about the same effect on visibility as the doubling of the aerosol concentration at constant humidity. Under real conditions, the reduction of visibility caused by precipitation must also be considered; this additional reduction of visibility (also shown in Figure 3-7) is meteorologically correlated with the relative humidity. Only a sufficient knowledge of the interaction of the natural and anthropogenous effects determining visibility will ultimately provide the criteria which may serve as bases for optimal--namely climate dependent (perhaps also weather dependent)--measures for limiting the aerosol concentration.

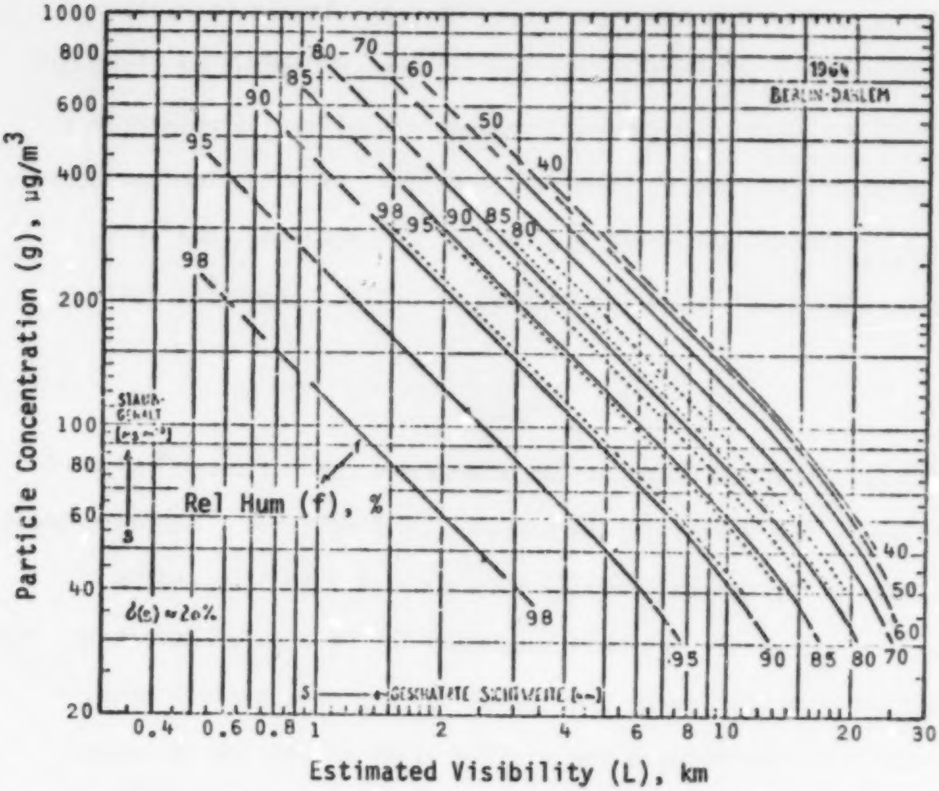

Figure 3-7. Relationship between visibility and particle concentration in conjunction with relative humidity in Berlin-Dahlem, derived from diurnal mean values 1964.[22] (The solid curves indicate the range measured values; the dotted curves were derived for precipitation-free cases.)

G. SUMMARY

The visual range, sometimes called the "visibility," is reduced by particulate matter in the air. It refers to the distance at which it is just possible to perceive an object against the horizon sky. Both the attenuation of light from the object and illumination of the air between the object and the observer tend to reduce visibility, since they reduce the perceived contrast between the object and its background. Attenuation of light passing through the air results from two optical effects which air molecules and small particles have on visible radiation: (1) the absorption of light energy and (2) the scattering of light out of the incident beam. In general, reduced visibility is primarily a result of scattering due to particulate matter. The "extinction coefficient," b_{scat}, is a measure of the degree of scattering, and it is related to the visual range (in meters) of a black object as follows:

$$L_v = \frac{3.9}{b_{scat}} . \qquad (3\text{-}3)$$

Suspended particulates found in the atmosphere cover a broad range of size; however, the visibility is affected by a relatively narrow segment of this size distribution, usually from about 0.1 to 1 μm radius. Once particulate matter has been suspended in the air for some time, the distribution of particles by size tends to take on a typical pattern. Because of this, and because the visible light scattering is caused primarily by particles of one narrow size range, the scattering can be empirically related to the particulate concentration. This relationship is as follows:

$$L_v \approx \frac{A \times 10^3}{G'} \qquad (\text{from } 3\text{-}9)$$

where G' is the particle concentration in μg/m^3, L_v is the equivalent visual range, and A is $1.2^{2.4}_{0.6}$ for L_v expressed in kilometers and $0.65^{1.3}_{0.3}$ expressed in statute miles. The ranges that are shown for the constant, A, cover virtually all cases studied. Deviations from equation 3-9 would be expected to occur when the relative humidity exceeds 70 percent, since many particles exhibit deliquescent behavior and grow into fog droplets. Particles composed of sodium chloride, for example, would act as condensation nuclei and show rapid and large changes in size under such circumstances. Also, this relationship may not hold for photochemical smog, since it is not known whether its size distribution conforms to the necessary pattern.

Equation 3-9 provides a convenient means for estimating the expected visibility for different levels of particulate concentrations, under the conditions stated. With a typcial rural concentration such as 30 μg/m^3, the visibility is about 40 km (25 statute miles); for common urban concentrations such as 100 and 200 μg/m^3, the visibility would be 12.0 km (7.5 statute miles) and 6.1 km (3.8 statute miles), respectively. In addition to aesthetic degradation of the environment, reduced visibility has many consequences for the safe operation of aircraft and motor vehicles. When airports have heavy traffic, visibility below 8.0 km (5 statute miles) tends to slow operations, since it is necessary to maintain larger distances between aircraft. Most air regulations prescribe limitations on aircraft operating under conditions of reduced visibility; they become increasingly severe as the visibility decreases from 8.0 km (150 μg/m^3) to 4.8 km (250 μg/m^3) to 1.6 km (750 μg/m^3). Based on the empirical variations in equation 3-9, the same visibilities could occur under certain circumstances, at concentrations one-half of these calculated values. Thus, a concentration of 75 $\mu g/m^3$ might produce a visibility of 8.0 km.

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Chapter 4

EFFECTS OF ATMOSPHERIC PARTICULATE MATTER ON MATERIALS

A. INTRODUCTION

Airborne particles may produce objectionable effects merely by settling on surfaces. A light deposit of dust makes surfaces appear dingy, and the frequent cleaning necessary in a dusty atmosphere weakens materials and is costly.

Particles can also cause direct or chemical deterioration of materials. The nature and extent of the deterioration depend on the chemical activity of the particles in their environment and the relative susceptibility of the receiving material. Particles may cause chemical deterioration either by acting as nuclei for the retention of sorbed gases or harmful acids, or by their own corrosive action.

The following sections will show that particulate air pollution plays an important role in the corrosion of metals; in the soiling, damage, and erosion of coated or painted surfaces, building stones, marble, and other building materials; and in the soiling and degradation of textiles.

B. EFFECTS OF PARTICULATE MATTER ON METALS

Atmospheric particles may accelerate the corrosion of iron, steel, and various nonferrous metals.

Metals are generally resistant to attack in dry air,[1] and even clean moist air does not cause significant corrosion.[1,2] Furthermore, inert dust and soot particles without sulfur compounds as constituents do not of themselves cause marked corrosion.[1,3] Particles may, however, contribute to accelerated corrosion in two ways.[4] First, they may be intrinsically active, and secondly, although inactive, they may be capable of sorbing active gases (such as SO_2) from the atmosphere.

Those particles that are inactive and have negligible capacity for sorption have little effect on materials. For example, silica particles do not accelerate the rate of metal corrosion even in the presence of SO_2. On the other hand, charcoal (carbonaceous) particles in atmospheres with relative humidities below 100 percent cause a large increase in the rate of corrosion in the presence of SO_2 traces, presumably through the local concentration of the gas by adsorption.[4] The laboratory research that led to these findings was based on particle concentrations of 0.4 mg/cm^2 (equivalent to 11.4 $tons/mi^2$) and abnormally high SO_2 concentrations of 270 mg/m^3 (100 ppm).

Active hygroscopic particles such as sulfate and chloride salts and sulfuric acid aerosols serve as corrosion nuclei. Their presence in the atmosphere can initiate corrosion, even at low relative humidities.[5] Laboratory studies of bare and varnished steel test panels, properly polished and degreased and then inoculated with finely divided particles of various substances commonly found in the atmosphere, were conducted by Preston and Sanyal.[5] Particles of chloride, sulfate, chromate, and oxide salts, and boiler and flue dusts were used. The metal test panels were exposed to atmospheres of clean air and of air containing SO_2 at various humidities, and the resulting corrosion was measured. Filiform corrosion, characterized by a filamental configuration, the primary phase in electrolytic corrosion, was noted in all cases. Corrosion rates are low when the relative humidity is below 70 percent, but they increase at higher humidities.[4,6] In most of the cases in this study, corrosion increased with increased humidity even in clean air. The addition of traces of SO_2 to the test atmosphere greatly increased the rate of corrosion in all instances.[5]

Field experiments show that the rate of corrosion of various metals is accelerated in urban and industrial areas because of the greater atmospheric concentrations of both particulate matter and sulfur compounds. Standardized open-hearth steel specimens, after exposure for one year at a number of diverse locations throughout the world, were observed by Hudson[7] to have a manifold variation in weight loss. Steel specimens exposed for one year to desert and arctic environments, the least polluted areas, were the least corroded; that is, they had the smallest losses in weight. Those specimens exposed at heavily polluted urban industrial sites were the most corroded, more so than similar specimens exposed at many marine and tropical locations, Table 4-1.

Table 4-1. CORROSION OF OPEN-HEARTH STEEL SPECIMENS IN DIFFERENT LOCATIONS[7]

Location	Type of atmosphere	Annual weight loss, g	Relative corrosivity
Khartoum, Sudan	Dry inland	0.16	1
Abisko, North Sweden	Unpolluted	0.46	3
Aro, Nigeria	Tropical inland	1.19	8
Singapore, Malaya	Tropical marine	1.36	9
Basrah, Iran	Dry inland	1.39	9
Apapa, Nigeria	Tropical marine	2.29	15
State College, Pa., USA	Rural	3.75	25
Berlin, Germany	Semi-industrial	4.71	32
Llanwrtyd Wells, British Isles	Semi-marine	5.23	35
Calshot, British Isles	Marine	6.10	41
Sandy Hook, N.J., USA	Marine-semi-industrial	7.34	50
Congella, South Africa	Marine	7.34	50
Motherwell, British Isles	Industrial	8.17	55
Woolwich, British Isles	Industrial	8.91	60
Pittsburgh, Pa., USA	Industrial	9.65	65
Sheffield Univ., British Isles	Industrial	11.53	78
Derby South End, British Isles	Industrial	12.05	81
Derby North End, British Isles	Industrial	12.52	84
Frodingham, British Isles	Industrial	14.81	100

Hudson, Figure 4-1, also correlated the rate of rusting of mild steel specimens and dustfall levels for four diverse areas, from a heavily industrialized area in Sheffield, England, to a rural area (Llanwrtyd Wells). The steel specimens corroded four times as fast in the polluted industrial atmosphere as they did in the rural atmosphere.

Committee B-3 of the American Society for Testing and Materials (ASTM)[8,9] studied corrosion rates of steel and zinc panels exposed for one year at several locations in the United States. The corrosivities of atmospheres at 19 sites were compared to that of the rural site of State College, Pennsylvania. This study [8] (Table 4-2) confirms Hudson's observations that industrial locations with high concentrations of particles and oxides of sulfur are more corrosive to steel and zinc than less industrialized areas. As shown in Table 4-2, steel specimens

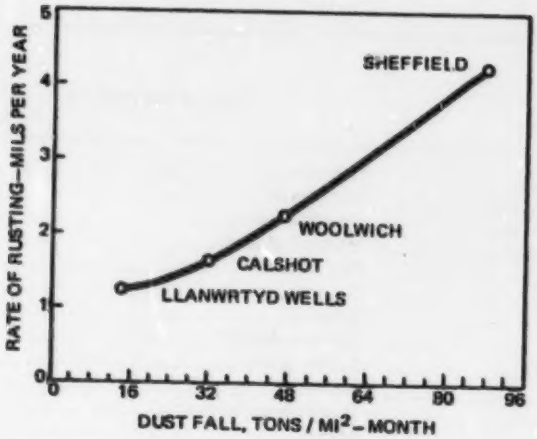

Figure 4-1. Plot of rate of rusting versus dustfall at four locations.[7] (The figure plots the rate of rusting of mild steel versus dustfall, and shows that corrosion is four times as rapid in an industrial area (Sheffield) as it is in the rural area (Llanwrtyd Wells)).

corroded in one year approximately 3.1 times as much in New York City (spring exposure) and 3.3 times as much in Kearny, New Jersey, as similar specimens at State College, Pennsylvania. Zinc specimens corroded 3.6 and 2.6 times as much in New York City (spring exposure) and Kearny respectively, as similar specimens exposed at State College. Both steel and zinc specimens in New York City (fall exposure when the particle and SO_2 concentrations in the atmosphere are greater than in spring) corroded 6.0 and 3.7 times as much respectively as similar steel and zinc specimens at State College.[8] The authors of the resulting papers did not give mean concentrations of suspended particles for the various locations; however, based on NASN measurements[10] made after the corrosion studies

Table 4-2. CORROSIVITY OF ATMOSPHERES TOWARDS STEEL AND ZINC AT SELECTED LOCATIONS RELATIVE TO THAT AT STATE COLLEGE, PENNSYLVANIA[8]

Location	**Type of atmosphere**	**Relative corrosivity of one-year test for**	
		Steel	**Zinc**
Norman Wells, N.W.T., Canada	Polar-Rural	0.03	0.4
Esquimalt, Vancouver Is., Canada	Rural-Marine [a]	0.5	0.4
Saskatoon, Sask., Canada	Rural	0.6	0.5
Perrine, Fla	Rural	0.9	1.0
State College, Pa	Rural	1.0	1.0
Ottawa, Canada	Semi-Rural	1.0	1.2
Middletown, Ohio	Semi-Industrial	1.2	.9
Trail, B.C., Canada	Semi-Rural	1.4	1.6
Montreal, Que., Canada	Industrial	1.5	2.2
Halifax, N.S., Canada	Rural-Marine	1.5	1.6
South Bend, Pa	Semi-Rural	1.5	1.5
Kure Beach, N.C., 800-ft site	Marine (800 ft from ocean)	1.8	1.7
Point Reyes, Calif	Marine	1.8	1.8
Sandy Hook, N.J	Industrial-Marine	2.2	1.6
New York, N.Y. (spring exposure)	Industrial	3.1	3.6
Kearny, N.J	Industrial	3.3	2.6
Halifax, N.S., Canada	Industrial-Marine [b]	3.8	18.0
New York, N.Y. (fall exposure)	Industrial	6.0	3.7
Daytona Beach, Fla	Marine	7.1	2.6
Kure Beach, N.C., 80-ft site	Marine (80 ft from ocean)	13.0	5.7

[a] While test site is 1500 ft. from brackish water, prevailing winds are from inland and prevent deposition of salt water spray.

[b] Test site is near a smokestack; prevailing winds blow fumes over the test site.

mean suspended particle concentrations were 176 $\mu g/m^3$ in New York City and 131 $\mu g/m^3$ at Elizabeth, New Jersey, near Kearney.[10] A town the size of State College, Pennsylvania, could have a mean suspended particulate matter level of 60 to 65 $\mu g/m^3$. It should be pointed out that there are significant differences in levels of gaseous pollution, particularly sulfur dioxide, between State College and the larger industrial communities. Consequently, the differences in corrosion rates cannot be solely attributed to the effects of particulates.

In Chicago and St. Louis, steel panels were exposed at a number of sites, and measurements were taken of corrosion rates and levels of sulfur dioxide and particulates.[11] In St. Louis, except for one exceptionally polluted site, corrosion losses correlated well with sulfur dioxide levels, averaging 30 to 80 percent higher than corrosion losses measured in nonurban locations. Over a 12-month period in Chicago, the corrosion rate at the most corrosive site (mean SO_2 level of 0.32 mg/m^3 or 0.12 ppm) was about 50 percent higher than at the least corrosive site (mean SO^2 level of 0.08 mg/m^3 or 0.03 ppm). Sulfation rates in St. Louis, measured by lead peroxide candle, also correlated well with weight loss due to corrosion. Although suspended particulate levels measured in Chicago with high-volume samplers correlated with corrosion rates, a covariance analysis indicated that sulfur dioxide concentrations had the dominant influence on corrosion. Measurements of dustfall in St. Louis, however, did not correlate significantly with corrosion rates. Based on these data, it appears that considerable corrosion may take place (i.e., from 11 to 17 percent weight loss in steel panels) at annual average sulfur dioxide concentrations in the range of 0.08 to 0.32 mg/m^3; and although high particulate levels tend to accompany high sulfur dioxide levels, the sulfur dioxide concentration appears to have the more important influence.

Comparative studies of the rates of corrosion of zinc-coated steel panels exposed to various community atmospheres were conducted by Committee B-3 of the ASTM.[9] The corrosion rates of zinc at four of the locations over a 6-year period are shown in Figure 4-2, as weight loss versus time.[8] For each location, the corrosion rate is essentially constant with time. The atmosphere of New York City, with greater concentration of particles and sulfur oxides, produces a more rapid rate of corrosion of zinc than that of Kearny, New Jersey, which is also heavily polluted.

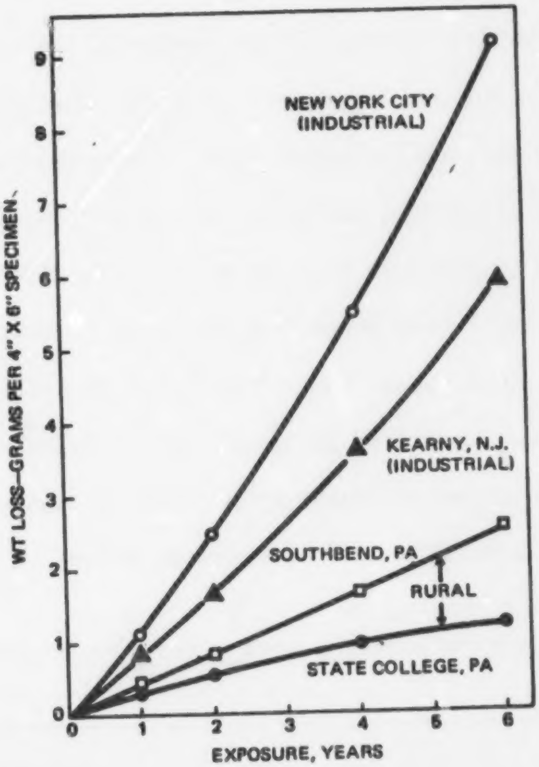

Figure 4-2. Weight loss from zinc specimen as a function of exposure time.[8] (The figure shows the rate of corrosion of zinc at four locations in the United States and indicates that corrosion is more rapid in industrial areas.)

Zinc corrodes at a greater rate in both industrial communities than in the rural or semirural sites in South Bend, Pennsylvania, and State College, Pennsylvania.[8]

Studies of the effects of air pollution on the atmospheric corrosion of three metals (nickel 200, Monel alloy 400, and copper) exposed to four diverse atmospheres (heavy industrial, urban-industrial, rural-farm, and rural-semiarid) were conducted over a 20-year period.[9] Nickel 200 is 99.5 percent nickel, and Monel 400 is essentially 30 percent copper and 70 percent nickel. Though these metals are relatively corrosion resistant, it is shown in Table 4-3 that they corroded more in industrial atmospheres than in rural ones. However, because of the corrosion resistance of the metals, all of the rates are relatively low when compared to those for unalloyed iron or steel. In the industrial locations, nickel 200 corroded at rates 2 to 4 times greater than Monel (nickel-copper) alloy 400, while in the rural locations, its corrosion rate was about equal to that of Monel alloy 400, and only one-half the rate for copper. Even Monel 400, with its superior corrosion resistance, when used as a gutter in an industrial installation, became pitted when the soot was permitted to accumulate.[9] Presumably, unburned carbon in the soot led to the formulation of local galvanic cells. The result was premature perforation and accelerated attack of the metal sheet.

Table 4-3. THE CORROSION RATES OF METALS IN VARIOUS LOCATIONS[9]

Test Site	Corrosion rate, mil/year		
	Nickel 200	Monel alloy 400	Copper
Altoona, Pa. (Heavy industrial-R.R.)	0.222	0.076	0.055
New York, N.Y. (Urban-industrial)	0.144	0.062	0.054
State College, Pa. (Rural-farm)	0.0085	0.007	0.017
Phoenix, Ariz. (Rural-semiarid)	0.0015	0.002	0.005

Larrabee[12] confirmed that the corrosion resistance of steel is greatly improved by the addition of small amounts of copper, or low alloys of chromium, nickel, copper, and phosphorus. The additional cost of such alloy steels when they are used to resist atmospheric corrosion is attributable to air pollution.

Particles can cause corrosion of electronic gear of all types even where precious metals are used to minimize such corrosion. Electrical instruments and electronic components are factors of growing importance in the computer and missile industries, and also monitor and control an increasingly large share of manufacturing processes.[13] Oily or tarry particles, commonly found in industrial and urban areas, are serious factors in the corrosion and failure of electric contacts, connectors, and components since they can produce a film with an affinity for dust.[13]

Dust can act mechanically or chemically on electric contacts. It can deposit on the surfaces and interfere with electrical contact closure, it can become imbedded in the surfaces of contacts, or it can induce wear by abrasion if the contacts slide.[13,14] Hygroscopic dusts, accumulated on contacts will absorb water to form thin electrolytic films which are corrosive to base metals.[5] If the contact members are not of identical composition, galvanic corrosion may occur. The tarnish and corrosion films impair or cause failures in electrical conductivity, and can be avoided or minimized only be fabricating the electrical contacts out of nonreactive metals, by encapsulation, or by air purification devices such as filters, or by gas-absorbing chemicals. Any of these solutions to the problem increases costs.

C. EFFECTS OF PARTICULATE MATTER ON BUILDING MATERIALS

Building materials and surfaces are soiled, disfigured, and damaged by atmospheric particles. Some of these stick to surfaces of stone, brick, paint, glass, and composition materials, forming a film of tarry soot and grit which may or may not be removed by the action of the rain. The result is a dingy, soiled appearance, a loss in aesthetic attractiveness and, in many cases, a physical-chemical degradation or erosion of these surfaces.

In cities where large quantities of soot-producing fuel are burned, the soiling problem is particularly severe. Much money and effort have been spent on sandblasting off the sooty layers which have accumulated on prominent buildings.[15,17] In Washington, D.C., it was found necessary to clean the soot and grime from the new Supreme Court Building even before initial occupancy (in the mid-thirties).[16] The tarry substances or carbonaceous material resulting from inefficient combustion of fuel are likely to be sticky and also acidic,[2] if not flushed off by rain they will adhere to surfaces and corrode them over extended periods of time.[18]

While the soiling effect of soot is by far the most evident to the eye, it does not in itself cause the deterioration of building material. The destruction is due to acids alone or to acids adsorbed on particulate matter.

D. SOILING AND DETERIORATION OF PAINTED SURFACES

Painted surfaces, walls, and ceilings of homes and other buildings are soiled by particles in the atmosphere. Furthermore, there are both liquid and solid particles present in polluted air in the form of fumes and mists of varying chemical composition that may react with painted surfaces.[18,19] The damage done is both aesthetic and material, and may affect not only buildings but also paintings and other works of art.

Auto finishes have been observed to become pitted and stained by iron particles deposited on them from a metal grinding operation nearby,[6,20] while chromic acid mist from an electroplating operation formed a brown stain on light-colored cars and a "blushing" on darker shades of paint. Repainting was required because the color changes were not reversible by washing or polishing.[18,21] Cars parked near demolition operations of brick buildings have been severely damaged by alkaline mortar dust in the presence of moisture.[18] Characteristic pitting of painted surfaces is also caused by sodium carbonate particles from such indus-processing as soda ash manufacture.

Water soluble chlorides and sulfates--mostly the iron, copper, calcium, and zinc salts--are commonly found in particulate samples from cities[19,21,22] and in rainwater.[21] These water soluble particles are potential sources of osmotic blistering of painted surfaces. The effects on weathering by small quantities of such particles have been examined[19] during laboratory studies of accelerated aging of various paint panels. The presence of 0.1 mg/l (0.1 ppm) of iron in the water in the accelerated weather apparatus produced yellow staining, while 0.5 mg/l (0.5 ppm) of copper produced severe brown staining. These same effects may be expected on natural exposure.[19]

Dust particles settling on wet varnish and paint films produce visible imperfections and reduce the electrical resistance and anti-corrosive properties of such films.[23] In laboratory tests to examine the relationship to corrosion of dust particles under the protective coatings, two sets of metal panels were coated with a varnish formulation under a "dust-free" apparatus, and then one set was removed from the apparatus so that the varnish films dried in contact with the laboratory air. The panels were then immersed in 3 percent aqueous sodium chloride for 7 1/2 months. Considerable rusting occured on the panels dried in "normal" laboratory room air after 6 months of immersion; no corrosion occured on the panels dried in the 'dust-free' environment. The dust particles that settled on the films of the panels during the drying period apparently were a factor in initiating corrosion of the metal.[23]

The exteriors of buildings in industrial areas, when repainted, collect numerous particles on their surfaces even before the paint has dried.[24] These particles produce imperfections in the protective surface and shorten its effective life.

E. SOILING AND DEGRADATION OF TEXTILES

Soiling of clothing, curtains, and other textiles not only diminishes their aesthetic appeal but also reduces their functional effectiveness. A garment which soils readily will not have the same user appeal as one which does not, even though its performance may be equal in other respects.

The soiling of a textile is influenced by various external factors such as temperature, relative humidity, and windspeed and the size and specific characteristics of the atmospheric particles. In addition, the degree of soiling is dependent on the construction and finish of the textile material and the type of fiber from which it is made.[25]

When large particles are deposited on the surface of the textile, the soiling may be superficial. In moving air, however, the material behaves as a filter. Laboratory studies by Rees,[25] using a dust-circulating apparatus under controlled conditions, show that a closely woven fabric of low porosity best resists soiling by airborne particles.

If an exposed textile material acquires an electrostatic charge, for example by friction, and retains this charge, charged particles of opposite sign will be attracted to it, thereby increasing its rate of soiling.[25] Some of the synthetic textiles acquire, by friction during spinning and weaving, electrostatic charges which, because of the high insulating properties of these materials, are retained for a long period of time. This results in troublesome "fog-marking," caused by attraction of airborne particles to the charged textile.[25]

In laboratory investigations of the electrostatic attraction of airborne particles to cotton textiles, Rees[25] found that soiling is more rapid when the fabric is charged than when uncharged, and that the rate of soiling is increased by raising the applied electric potential (which increases the charge density on the fabric). For any given potential, the rate of soiling appears to be greater for a positively charged fabric specimen than for a negatively charged one.

The vulnerability of textile fabrics and furnishings to acid components of particulate matter depends on the chemical composition of the textiles.[26] Cellulosic fibers, such as cotton, linen, hemp, jute, and manmade rayon, are particularly sensitive to attack from such substances as sulfurous and sulfuric acids, while animal fibers, such as wool and furs, are more resistant to acid damage.[26]

Curtains are particularly vulnerable to air pollutants and often deteriorate quickly hanging at open windows. The curtain material acts more or less as a filter for particles. Airborne metallic iron and zinc particles, constituents of city dust,[21,26,27] are catalysts and promote oxidation of sulfur dioxide to sulfuric acid, which may contribute to textile degradation. Curtains weakened by conditions of exposure arising partly from atmospheric soiling and acidity give way in a characteristic manner by splitting in parallel lines along the folds where the greatest number of particles accumulated.

F. SUMMARY

Airborne particles--including soot, dust, fumes, and mist--can, according to their chemical composition and physical state, cause a wide range of damage to materials. They may cause deterioration merely by settling on surfaces and soiling them, thus creating a need for more frequent cleaning which in itself weakens materials. More importantly, they can cause direct chemical damage to materials in two ways: First, through their own intrinsic corrosiveness, and secondly, through the action of corrosive chemicals which they may have absorbed or adsorbed. Airborne particulates have been implicated in the corrosion of metals and metallic surfaces; in the soiling, damage, and erosion of buildings and other structures; in the discoloration and destruction of painted surfaces; and in the aesthetic degradation and damage of fabrics and clothing.

Metals ordinarily can resist corrosion in unpolluted dry air, or even in moist clean air. Even inert dust or soot, in the absence of active chemical agents, has little effect on metal surfaces. However, hygroscopic particles commonly found in the atmosphere can corrode metal surfaces although no other pollutants may be present. This was shown in laboratory studies in which steel test panels were dusted with various common particulates. Although corrosion rates were low at relative humidities under 70 percent, they tended to increase with increased humidity. The addition of sulfur dioxide to the laboratory air greatly accelerated the rates of corrosion.

In general, the rate of corrosivity increases as we go from dry, unpopulated environments, the least polluted areas, to heavily polluted urban industrial sites. Samples of iron, for example, have shown weight losses due to corrosion that are four times greater in industrial atmospheres than in rural atmospheres. Steel samples corroded, in 1 year, 3.1 times as much in New York City (spring exposure), where the particulate concnetration was 176 $\mu g/m^3$, as in the rural atmosphere of State College, Pennsylvania, where the mean concentration could be expected to range about 60 to 65 $\mu g/m^3$. Steel samples exposed in New York in the fall of the year, when the particulate and sulfur dioxide levels are higher than in the spring, corroded 6 times faster than the samples at State College. Similarly, zinc samples exposed in New York corroded about 3.6 times as much as those at State College, while zinc samples at Kearny, New Jersey, corroded about 2.6 times as much (the concentration was 131 $\mu g/m^3$ at Elizabeth, New Jersey, near Kearney).

It should not be concluded, however, that the variation in corrosion rates reffered to above is due only to differences in particulate matter, since significant differences in gaseous pollutant concentrations existed between State College and the other areas. Even highly resistant metals have shown corrosion rates that are progressively larger over the following range of environments: (1) a rural-semiarid site, (2) a rural-farm environment, (3) an urban-industrial area, and (4) a heavy industrial-R.R. area. In Chicago and St. Louis, steel panels were exposed at a number of sites, and measurements were taken of corrosion rates and of levels of sulfur dioxide and particulates. In St. Louis, except for one exceptionally polluted site, corrosion losses correlated well with sulfur dioxide levels, averaging 30 to 80 percent higher than corrosion losses measured in nonurban locations. Over a 12-month period in Chicago, the corrosion rate at the most corrosive site (mean SO_2 level of 0.32 mg/m^3 or 0.12 ppm) was about 50 percent higher than at the least corrosive site (mean SO_2 level of 0.08 mg/m^3 or 0.03 ppm). Sulfation rates in St. Louis, measured by lead peroxide candle, also correlated well with weight loss due to corrosion. Although suspended particulate levels measured in Chicago with high-volume samplers correlated with corrosion rates, a covariance analysis indicated that sulfur dioxide concentrations had the dominant influence on corrosion. Measurements of dustfall in St. Louis, however, did not correlate significantly with corrosion rates. Based on these data, it appears that considerable corrosion may take place (i.e., from 11 to 17 percent weight loss per year in steel panels) at annual average sulfur dioxide concentrations in the range of 0.08 to 0.32 $\mu g/m^3$ (0.03 to 0.12 ppm), and although high particulate levels tend to accompany high sulfur dioxide levels, the sulfur dioxide concentration appears to have the more important influence.

Particles play a significant role in corrosion and damage to electronic equipment of all kinds, even when precious metals are used to minimize such effects. The contacts in electrical switches are vulnerable to chemical or mechanical action by particulates. The particulates commonly found in industrial and urban atmospheres are serious factors in the corrosion and failure of electrical connectors and contacts.

The ability of particulates to damage and soil buildings, sculptures, and other structures is particularly great in cities where large quantities of coal and sulfur-bearing fuel oil are burned. Particles may cause deterioration to many types of masonry by acting as reservoirs for the harmful acids generated by the combustion of these fuels. In addition to direct erosion and physical degradation of materials, particles stick to surfaces with which they come in contact, forming a film of tarry soot and grit which may or may not be removed by the action of rain. The result is a dingy, soiled appearance, and much money and effort must be spent to sandblast off the sooty layers that accumulate.

Particles also may soil the painted surfaces of walls, ceilings, and the exteriors of homes and buildings, and, under certain circumstances, may cause them to become stained and pitted. Automobile finishes, for example, have been damaged by particulate matter emitted from nearby industrial operations. Water soluble chlorides and sulfates--mostly the iron, copper, calcium, and zinc salts--are commonly found in particulate samples from cities and in rainwater and may cause blistering and enhance the weathering of painted surfaces. Particles may settle on painted surfaces before the paint has dried, thus producing imperfections and reducing the ability of the paint to protect the surface. Such surfaces are likely to soon require refinishing.

The soiling of clothing, curtains, and similar textiles makes them unattractive and diminishes their use. The extent of soiling is influenced by a number of factors, including temperature, relative humidity, and air movement. Small particles may penetrate deep into the fibers of fabrics exposed to moving air. The vulnerability of textile products to the acid components of airborne particles also depends on the composition of the material. Cellulosic fibers, for example, such as cotton, linen, hemp, jute, and man-made rayon, are particularly sensitive to attack from such substances as sulfurous and sulfuric acids. In addition to the aesthetic degradation and the nuisance created by particulate matter, direct costs may be associated with the increased rate of deterioration of textiles, the extra cleaning required, and the manufacturing expenses for fabrics that are more resistant to air pollution.

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Chapter 5

ECONOMIC EFFECTS OF ATMOSPHERIC PARTICULATE MATTER

A. INTRODUCTION

It is perhaps, misleading to cordon off the so-called economic effects of particulate pollutants from the effects which are the foci of this document. All of the effects discussed in this document have economic dimensions although some are easier to measure than others. For example, it is widely believed that human health transcends economic value; yet, in fact, there is a finite value even though it is not measured easily.

Difficult or easy, such valuation is a necessary part of the air quality standards decision process. Communities seeking a more favorable environment are confronted with the need for two bodies of fact. First, there are the physical laws, technological factors, and economics that govern the generation, transport, and control of air pollution. Second, there are the undesirable effects of pollution on man and his environment. These two realities conspire to produce a dilemma. This dilemma arises from the two sides of the economic aspect of air pollution: (1) air pollution causes damage and losses which must be borne by the community, and (2) the prevention of these damages (or the reduction in air pollution) involves using resources which could be used for other worthy community objectives. It is evident that some sort of balance must be struck. Precisely where this balance can be achieved is a matter for each affected community to decide, but economics will play a role.

The tools of economic analysis can illuminate the nature of this dilemma and, with some qualifications, can even provide solutions. It is sometimes suggested, for instance, that the monetary value of the undesirable consequences of not controlling air pollution be balanced against the consequences of instituting control. This is the objective of the much-heralded cost-benefit analysis, which provides the decision-maker with alternatives from which he may choose that which seems most favorable. Comparisons are made, insofar as possible, in monetary terms, with the coin of the realm serving as a common denominator. It is the purpose of this chapter to review the growing body of literature on the economic value of the effects considered in other chapters of this volume.

A major difficulty in assessing damages resulting from particulate air pollution lies in isolating the effects of particles, per se. At present, technology is inadequate to permit ready determination of the effects of individual air pollutants acting synergistically with other pollutants or of the monetary cost of these effects. Increased attention, including expanded research effort, must be given to developing this technology.

Another problem is that an individual's consumption pattern is a function of numerous socioeconomic variables, and the assessment of the marginal impact of one variable, particulate air pollution, requires a number of assumptions. More sophisticated analytical techniques, heretofore not used in the economic analysis of pollution effects, are being used to better define cause-and-effect relationships. Such techniques include, among others, input-output and general equilibrium models.

A major objective of research into the economic effects of air pollution is to establish quantitative relationships between varying levels of pollution and economic damages--the economic damage function.

In turn, when such damages are averted, this economic damage function estimates the benefits accrued from air pollution control.

B. METHODS FOR MEASURING AIR POLLUTION DAMAGES[1,2,3]

In constructing the benefit side of a cost-benefit analysis for air pollution abatement, we would want to know how much consumers would pay for abatement if the economic world were optimal except for air pollution. There are several approaches that can be used to derive this demand schedule.

The first is called the differential site value or property value method. This method assumes that consumers consider reduced air pollution to be a benefit and will act as if they know the value of this benefit. Prices, such as those of residential property, will reflect this valuation of the benefit. The property market is more likely than others to reflect the majority of the residual effects of air pollution which represents losses in human welfare and can serve as surrogate for many of the various direct and indirect negative impacts of adverse environmental changes.

The second approach focuses on the application of an opinion survey. If it can be assumed that consumers know explicitly the benefits of pollution abatement, then the objective must be to elicit complete information from him in a way that would dissuade lying. This approach is closest to the classical economic approach in that it focuses on estimating utility and demand functions. Questionnaire interview studies have demonstrated their ability to obtain information on perception of air pollution and attitudes toward it.[4,5,6] Interviews can provide the investigator with information about the type, nature, and extent of air pollution effects. Such a scheme aids in the attachment of relevant economic values to those goods that are not traded in the market place, or more precisely, to what people would be willing to pay for the benefits resulting from cleaner air.

The third procedure uses the technical coefficients of production and consumption. This approach, which is the most easily handled of the three, has been most often employed.[7,8,9] Copious literature exists about the physical effects of air pollutants on organisms and objects. Experimental data can be derived from observations on economic objects in conditions corresponding closely to actual use. With this information, the physical damage function which relates physical damage to pollution levels can be estimated; these functions can then be translated into economic damage functions. Thus, measurements of either the direct losses or the expenditures made to avoid or offset such direct losses are made and extrapolated to the whole population or damage category.

Another method that appears to have potential but will not be discussed in depth here is the litigation survey. This method depends on the development of some way of deriving information on air pollution damages from the decisions of the judicial system on conflicts of interest over the air resource. Havighurst[10] attempted to estimate air pollution costs by surveying litigation records in Philadelphia and evaluating the judicial data as estimators of damage functions.

C. EARLY ECONOMIC STUDIES

One of the first comprehensive surveys of damage due to air pollution in the United States was conducted in Pittsburgh during the years 1912 to 1913 by the Mellon Institute.[11] The estimates in the Pittsburgh investigation were based on economic losses due to dustfall and smoke and were obtained through interviews of individuals. The items

Table 5-1. ESTIMATED COSTS DUE TO AIR POLLUTION (MELLON INSTITUTE PITTSBURGH STUDY)[11]

Causes of expenditure	Annual cost
To smokemaker:	
Imperfect combustion	$1,520,740
To individual:	
Laundry bills	1,500,000
Dry cleaning bills	750,000
To household:	
Exterior painting	330,000
Sheet metal work	1,008,000
Cleaning and renewing wall paper	550,000
Cleaning and renewing lace curtains	360,000
Artificial lighting	84,000
To wholesale and retail stores:	
Merchandise	1,650,000
Extra precautions	450,000
Cleaning	750,000
Artificial lighting	650,000
Department stores	175,000
To quasi-public buildings:	
Office buildings	90,000
Hotels	22,000
Hospitals	55,000
Total	$9,944,740

of cost and the total cost to the community were ascertained as shown in Table 5-1.

The Mellon Institute investigators arrived at an annual per capita cost of $20 in 1913. Health effects were not included; aesthetic losses at the time were judged to be $5 per person above the $20.[11]

A comprehensive study of the economic effects of air pollution in Great Britain was described in the Beaver Report in 1954.[12] This study considered both direct costs and efficiency losses. The direct costs included laundry and domestic cleaning; the cleaning, paint, and repair of buildings; the corrosion of metals and consequent cost of replacement and of providing protective coverings; damage to goods; additional lighting; and extra hospital and medical services. Efficiency losses were represented by the effects on agriculture of damage to soil, crops, and domestic animals; interference with transport; and reduced human efficiency due to illness.

Le Clerc, writing in 1961 about economic losses due to air pollution, cited data from France and Great Britain, as well as some general data on economic losses in the United States. The foreign data were quoted in local monetary units, which have varied over the years in relation to the value of the dollar. Even those figures relating to the United States are cited in the context of the value of the dollar at the time of study. Some of the data contained estimates on medical services; others did not.[13]

In these two studies no attempt was made to relate economic losses to the ambient particle concentration.

D. RECENT ECONOMIC STUDIES

1. Public Opinion Surveys

The public opinion, questionnaire type survey has been used on numerous occasions to evaluate the social concern about air pollution and willingness to pay for its abatement. Ridker[14] used this technique and surveyed the residents near a power plant to determine the additional cleaning costs resulting after a malfunction in the boilers caused fumigation of the neighborhood with unusually large amounts of soot. This survey determined the cost of cleaning up and the willingness-to-pay cost. The difference between measured cost and willingness-to-pay cost is the psychic cost--a loss beyond the direct, measured costs. The survey results indicate that the households generally were willing to pay, on the average, 27 percent above the measured cost of cleanup in order to avoid the cleanup.

These results must be qualified. Since the survey was constructed in only a few days, one must allow for the possibility of an incomplete questionnaire and an inadequate sample.

Another survey of willingness-to-pay was conducted in 1965 by Lawyer[15] in Morgantown, West Virginia. Table 5-2 shows what the respondents would pay each year "...if all air pollution were reduced below the point where it was noticeable (or harmful)."[15] The average

Table 5-2. AMOUNT OF MONEY RESPONDENTS WOULD PAY ANNUALLY TO REDUCE AIR POLLUTION IN MORGANTOWN, WEST VIRGINIA[15]

Amount, $	Respondents, %
Zero or no response	38.4
1-5	23.9
6-10	9.4
11-15	2.5
16-20	4.7
21-25	6.3
26-30	0.3
31-35	0.6
36-40	0.3
>40	13.5
	99.9[a]

[a] Error due to rounding.

amount that each respondent would be willing to pay annually was calculated to be \$16.46.

The average annual payment of \$16.46 for each respondent is much higher than the amount indicated by the willingness-to-pay study by Williams and Bunyard.[16] They reported that 66 percent of those interviewed in a 1963 survey of the St. Louis area was willing to pay \$5 a year in higher living costs for cleaner air and that 85 percent would pay \$1 a year in higher taxes.

2. Site Differential

The first serious use of this approach was made by Ridker and Henning.[17] Using 1960 census data and pollution measurements from a 1963-64 air pollution study, they explained over 90 percent of the variation in the median property values (MPV's) for small areas of the city at St. Louis, Missouri. A number of descriptive variables such as housing characteristics, population density, and other pertinent socioeconomic variables were included in the regression equation. The average value of the MPV changed by \$73 to \$417 for every 0.25 mg $SO_3/100\ cm^2$-day change in concentration of annual geometric mean sulfation levels.

Based on this method, Anderson and Crocker[18] studied the covariation of sulfation, suspended particles, and census tract MPV'S in St. Louis, in Washington, D.C., and in Kansas City. They obtained the highest precision by isolating the following variables:

a. Arithmetic mean sulfation rate;

b. Annual arithmetic mean suspended particulates;

c. Median family income;

d. Percentage homes dilapidated;

e. Percentage homes more than 20 years old in 1959;

f. Distance to central city.

They concluded that the MPV changes by 10 to 20 percent in response to variation in pollution levels. For every change of 0.1 mg SO_3/100 cm^2-day in sulfation and of 10 $\mu g/m^3$-day of suspended particles over a particular section of the city, the best estimate of the change in that section's MPV lies in the $300-to-$700 interval.

Zerbe, using similar methods, reports for Toronto "that, other things being equal, for each increase in 1 mg SO_3/100 cm^2-day property values will fall by an amount between $800 and $1,800 for each single family detached dwelling, and that the best estimate is that the values will fall by about $966."[19]

Jaksch and Stoevener[20] conducted a study in Toledo, Oregon, which used dustfall measurements. Their hypothesis was that air pollution represents an economic cost to the affected community and that such cost is reflected in the property values. Unique to this study is the use of actual sales data of residential property within the city limits to provide the empirical basis for testing the hypothesis. They found that reductions in property values from increasing air pollution levels are likely to be greater for higher valued, more developed properties than for less developed, lower valued properties. Two models were developed to analyze the relationship between dustfall pollution and residential property values. In one, it was found that an increase of 1 ton/mi^2-mo in dustfall caused property values to decline by $277 per acre; in the other, it was found that a similar increase in dustfall caused a decline in property values of $29 per market transaction.[20]

In an extension of the method developed by Ridker and refined by Anderson and himself, Crocker[21] recently completed a study which focused on the theory and measurement of urban air pollution damage functions. He concluded that the "best" estimate of average marginal capitalized air pollution damages in the city of Chicago is about $500 per single family residential unit for each additional 10 $\mu g/m^3$-day of suspended particulates plus 1 ppb per 72 hours of SO_2. In summary, Crocker said that "differential site values can be employed to obtain a lower bound on air pollution damages; and if the sites in question have rather homogenous characteristics, their differential values represent a substantial portion of damages."[22]

3. TECHNICAL COEFFICIENTS

Two studies have attempted to estimate the costs of air pollution on human health. Although particulate pollution was not isolated as the sole pollutant responsible for the measured effects, the role and significance of this variable in these studies is indisputable. Both follow the same general method used in technical coefficients studies: (1) an estimate is made of the value of total health losses for a particular disease (damage category), (2) a coefficient is determined for the share of this value attributable to air pollution, and (3) the product of total health losses times the coefficient yields the value of health losses associated with air pollution for that specific disease or illness.

The earliest published attempt at estimating the cost of health from air pollution is that of Ridker and Henning.[17] They estimated the total national cost of morbidity and mortality for diseases associated with the respiratory system. The cost estimates for each disease included the costs of premature death, premature burial, treatment, and absenteeism. Summing the costs for 1958 yielded the total economic cost for these diseases of $199 billion. By applying coefficients of 0.18 and 0.20 that were taken from other studies, the damage to health from air pollution in 1958 was estimated to be in the range of $360 and $400 million.

Ridker considers the estimate to be conservative because: (1) avoidance costs representing the moving costs and possible the reduced earnings of those who migrate to areas of lower pollution because of their health were not considered; (2) the value of housewife services lost due to death or illness related to air pollution was not imputed; (3) no attempt was made to include the psychic costs associated with death or illness; and (4) data limitations prevented the consideration of some diseases.

The Lave-Seskin[23] study expands the scope of diseases covered by Ridker. It included the costs of premature death, treatment, and absenteeism by major disease category for 1963. The coefficients for the diseases were estimated by regressions that were run on data from published epidemiological studies. In these, morbidity and mortality rates were expressed as functions of air pollution and selected socioeconomic variables.

The total annual cost of air pollution in increased human morbidity and mortality was estimated to be $2.08 billion for 1963; or inversely stated, a 50-percent reduction in air pollution would result in annual savings of $2.08 billion. Table 5-3 summarizes their results. Their preferred index is that the health cost of pollution amounts to 4.5 percent of all the economic damage associated with morbidity and

Table 5-3. ANNUAL HEALTH COST OF AIR POLLUTION[23]

Disease	US$ Millions
Respiratory	
Bronchitis	500
Other	722
Cancer	
Lung	33
Other	357
Cardiovascular	468
Total	2,080

mortality. Lave and Seskin's work suffers from many of the same difficulties as Ridker's in deriving meaningful estimates. However, it is felt that their results are reasonable. It should be pointed out that changes in health costs cannot be related to changes in indexes of pollution.

Another general damage category in which the technical coefficients method has been employed is that of household soiling. The primary purpose of soiling studies, in general, is to ascertain the relationship between cleaning frequencies and some measure of particulate air pollution.

Michelson and Tourin[24] have received more attention than other researchers in this field. They used a mail questionnaire in a study in Steubenville and Uniontown in the Upper Ohio Valley. A positive relationship was found to exist between the clean frequency and the particulate level. Per capita costs for Steubenville, with an annual average particulate level of 235 $\mu g/m^3$, were $84 greater than those of Uniontown, where the annual average particulate level was 115 $\mu g/m^3$.

To validate this procedure, Michelson and Tourin conducted a subsequent survey in the Washington, D. C., area.[25] Serious doubt has been cast upon their conclusions because income was the only controlled variable in the analysis and because of some basic differences between this and the Ohio studies. They also extended this methodology to estimate household soiling costs in Connecticut.[26]

A residential soiling study was conducted in Philadelphia in 1969 by Booz-Allen and Hamilton, Inc.[27] The survey was designed to remove some of the uncertainty generated by the earlier studies. The study concluded that certain low-cost cleaning and maintenance operations appear sensitive to particulate levels, but high-cost operations are relatively unaffected by variations in the particle concentrations in the Philadelphia area.

E. DISCUSSION

Costs derived by the use of the three methods discussed in this chapter involve overlapping estimates. Each technique has its own inherent strengths and weaknesses depending on the information available and the objectives of the investigator.

The problem of overlap in the estimates is but one problem in the assessment of pollution costs. Although this chapter reviews the costs of particulate air pollution, it is obvious that this pollutant does not exist alone in the atmosphere; many pollutants are always present simultaneously and numerous complex physicochemical reactions occur between them. For instance, gaseous sulfur dioxide goes through a chemical reaction whereby it oxidizes to sulfate, a particulate. Reactions such as this increase the difficulty of defining the cause-and-effect relationships needed to determine meaningful cost functions.

F. SUMMARY

Studies of air pollution and residential property values and health effects, while limited by data availability, strongly suggest that a statistically significant inverse relationship may exist between higher levels of particulate and other pollution and these effects.

The work by Lave and Seskin[23], and Anderson and Crocker[18] provide the most objective evidence of the economic magnitude of atmospheric particulate matter. Lave and Seskin estimate that a 50-percent reduction in air pollution (sulfation and particulate pollution concentrations) would have resulted in annual savings in costs of human morbidity and mortality of $2.08 billion in 1963.

Anderson and Crocker conclude that for every change of 0.1 mg/ SO_3/100 cm^2-day in sulfation and 10 $\mu g/m^3$-day of suspended particulates over a particular small section of an urban area, the best estimate of the change in that area's median property value is between $300 and $700. This is consistent with Crocker's results in his Chicago study in which he affirms that the "best" estimate of average marginal capitalized air pollution damages is about $500 per single family residential unit for additional 10 $\mu g/m^3$-day of suspended particulates plus 1 ppb of SO_2/72 hours.[21]

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Chapter 6

EFFECTS OF ATMOSPHERIC PARTICULATE MATTER ON VEGETATION

A. INTRODUCTION

Little is known about the effects of particulate matter on vegetation and little research has been done on the subject. There has been far more research on gaseous pollutants, many of which are readily recognized as serious toxicants to a variety of plants. Published experimental results mostly from Germany and Czechoslovakia, are confined principally to settleable dusts emitted from the kilns of cement plants. There are few reports on effects of fluoride dusts, soot, and fumes from certain types of metal processing. Sulfuric acid aerosols have also been studied in Los Angeles, where deposition of acid droplets has injured the leaves of vegetation. Most of the research was related to the direct effects of dusts on leaves, twigs, and flowers as opposed to indirect effects from dust accumulation in the soil. Because of the dearth of experimental results, the tenor of many reports is directed as much to the question "Do dusts in fact have deleterious effects on plants?" as to the question of extent of injury. Such information as there is refers to specific dusts rather than to the conglomerate that is usually measured as urban or rural dustfall (Chapter 1). The various specific pollutant dusts and their injurious effects on vegetation are discussed in the following sections.

B. EFFECTS OF SPECIFIC DUSTS ON VEGETATION

1. Cement-Kiln Dust

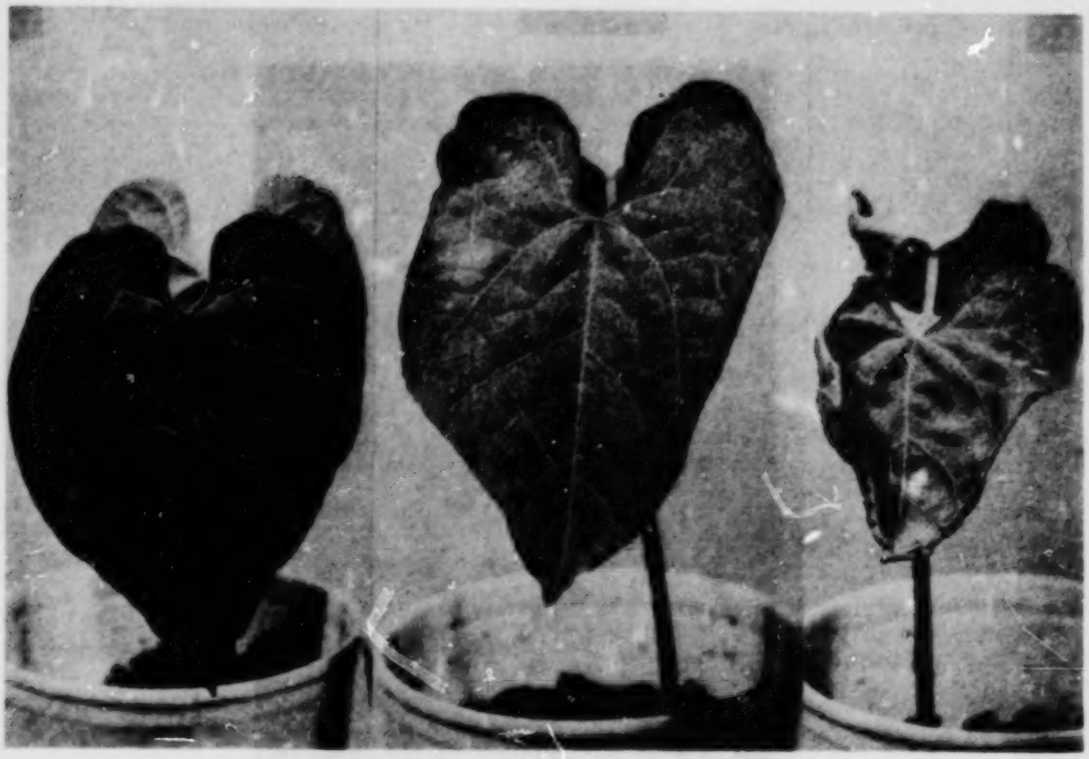
Cement-kiln dust is the dust contained in waste gases arising from the drying, caloining, and clinkering processes of the kilns. It is apparent from some reports, however, that the composition of wastes from different kilns operating at different efficiencies varies considerably, and at times the effluents may contain cementitious materials that more properly belong in the finished product. Another important factor to consider is that reports describing effects of dust deposited on various plants in the field relate to kiln-stack materials, whereas experimental dusts applied in laboratory or field studies were taken from various collectors in the waste-gas system between the kiln and the stack. Differences in results that may be due to this factor have not been identified.

a. Direct Effects

(1) Nature of Dust Deposition. Most of the reports concerning harmful effects of cement-kiln dust on plants stress the fact that crusts form on leaves, twigs, and flowers. As early as 1909-1910 Peirce[1] and Parish[2] noted in California that settled dust in combination with mist or light rain formed a relatively thick crust on upper leaf surfaces of affected plants. The crust would not wash off and could be removed only with force. The central theme about which Darley,[3,4] Czaja,[5] and Bohne[6] build their cases for harmful effects is the crust formation in the presence of free moisture. They state that crust is formed because some portion of the settling dust consists of the calcium silicates which are typical of the clinker (burned limestone) from which cement is made. When this dust is hydrated on the leaf surface, a gelatinous calcium silicate hydrate is formed, which later crystallizes and solidifies to a hard crust. When the crust is removed, a replica of the leaf surface is often found, indicating intimate contact of dust with the leaf. The relatively thick

crust formed from continuous deposition is confined to the upper surfaces of leaves of deciduous species but completely encloses needles of conifers. Prolonged dry periods during the time dust is deposited provide no opportunity for hydration, and crusts are not formed. Dust deposits which are not crusted are readily removed by wind or hard rain.

Darley[3] applied kiln dusts of particle size less than 10 μm at rates



a b c

Figure 6-1. Bean plants dusted with cement-kiln particles: (a) control plant; (b) dusted but kept dry; (c) exposed to natural dew formation after each day's dusting.[4] (The photograph shows the effect of dusting bean plants for 2 days with particles (8 to 20 μm) from a cement kiln. The plant exposed to moisture (natural dew) and dust is most affected. The dusting rate was 0.47 mg/cm^2-day.)

of 0.05 to 0.38 mg/cm^2-day to leaves for 2 to 3 days in the laboratory. Water mist was applied several times each day. Even though the dust adhered to the leaf in a uniform layer, it did not appear to be crustlike, probably because the experiments were of short duration. Reduction of CO_2 intake was reported in these [illegible]iments.

Leaves of bean plants dusted for 2 days wi[illegible]nt-kiln dust of 8 to 20 μm in size at the rate of 0.47 mg/cm^2-day, [illegible]atively high

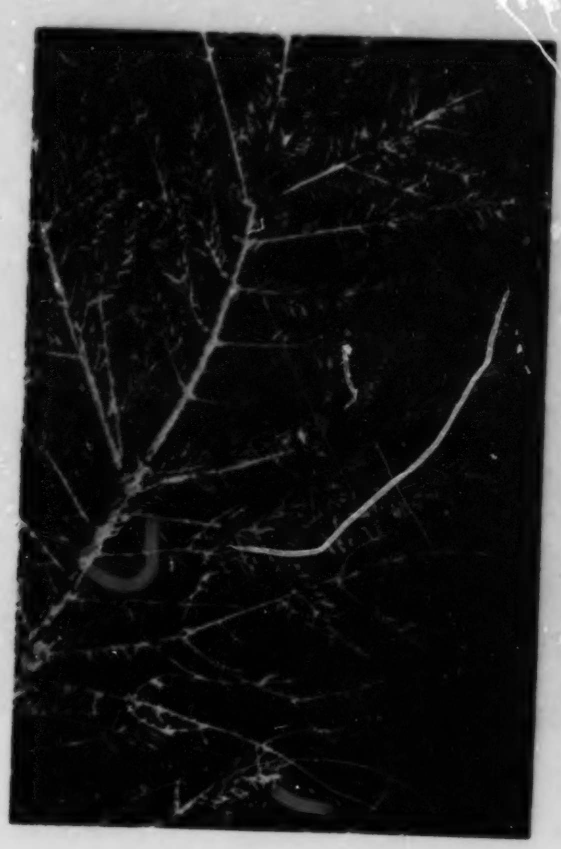


Figure 6-2. Cement-kiln dust on fir tree branches.[4] (Incrustation has built up on the older twigs of a fir tree exposed to a cement-kiln dustfall probably in excess of 0.1 mg/cm^2-day. Needles have fallen prematurely.)

rate, and then exposed to naturally occurring dew were moderately damaged.[4] The injury (Figure 6-1) appeared as a rolling of the margins of the leaves, and some interveinal tissue was killed. Leaves which were dusted but kept dry were not injured.

Photographs taken in Germany by Darley[4] (Figures 6-2 and 6-3) show incrustations of cement-kiln dust on branches of fir trees. Atmospheric levels of dust in this area were probably in excess of 0.1 mg/cm^2-day

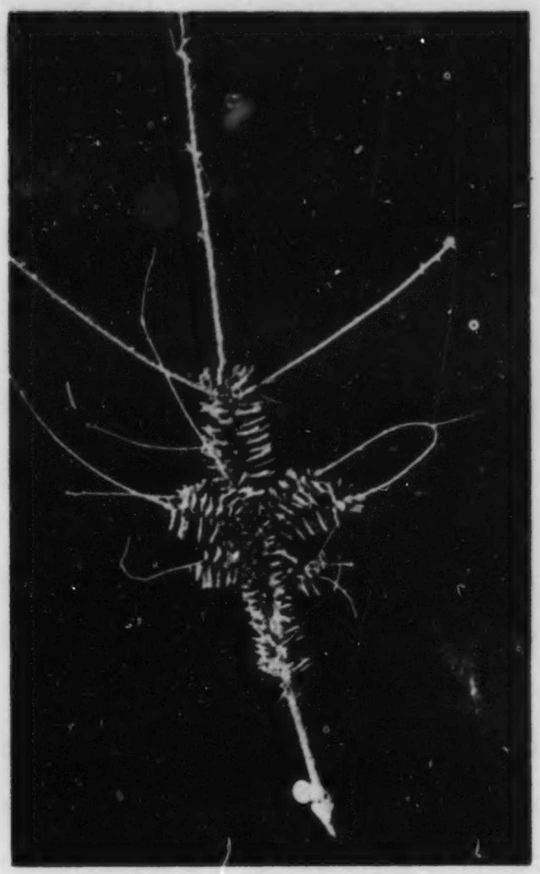


Figure 6-3. Cement-kiln dust on fir tree branches.[4] (Very heavy incrustation on a branch of a dead fir tree exposed to cement kiln dust (dustfall rates probably in excess of 0.1 mg/cm^2-day).)

(85 tons/mi^2-mo). Incrustations built up on the older twigs (Figure 6-2) and caused needles to fall prematurely. Some of the twigs were dead and incrustations were forming on the newest needles. The net effect was a shortening of each succeeding year's flush of growth. A dead tree had heavy incrustations on the branches (Figure 6-3).

(2) Range of Effects. Peirce[1] demonstrated that incrustations of cement-kiln dust on citrus leaves interfered with light required for photosynthesis and reduced starch formation. This was later confirmed by Czaja[5] and Bohne[6] in a variety of plants. More recently, Steinhubel[7] compared starch reserve changes in undusted common holly leaves and those dusted with foundry dust. He concluded that the critical factor in starch formation was the light absorption by the dust layer, and that the influence on transpiration or overheating of leaf tissue was of minor significance. Lecenier and Piquer (see Czaja[5]), attributed the reduced yields from dusted tomato and bean plants to interference with light imposed by the dust layer. Darley[3] demonstrated that dust deposited on bean leaves in the presence of free moisture interfered with the rate of carbon dioxide exchange, but no measurements of starch were made.

Czaja[5] stated the hydration process of crust formation released calcium hydroxide. The hydrated crusts gave solutions of pH 10-12. Severe injury of naturally dusted leaves, including killing of palisade and parenchyma cells, was revealed by microscopic examination. The alkaline solutions penetrated stomata on the upper leaf surface, particularly the rows of exposed stomata on needles of conifer species, and injured the cells beneath. Czaja[5] stated that on broad-leaved species with stomata only on the lower leaf surface, the alkaline solutions first saponified the protective cuticle on the upper surface, permitting migration of the solution through the epidermis to the palisade and parenchyma tissues. Typical alkaline precipitation reactions with tannins, expecially in rose and strawberry leaves, were evidence that calcium hydroxide penetrated the leaf tissue. Bohne[6] described similar "corrosion" of tissues under the crust formed on oak leaves.

Czaja[8] has presented good histological evidence that stomata of conifers may be plugged by dust, preventing normal gas exchange by the leaf tissue. Uninhibited exchange of carbon dioxide and oxygen by leaf tissue is necessary for normal growth and development.

Bohne[6] reported a marked reduction of growth of poplar trees located about 1.6 km (1 mile) from a cement plant after production in the plant was more than doubled. The change in growth rate was determined by the width of annual rings in the wood. Darley[3] observed a reduction of spring growth elongation on conifers in Germany, where the oldest needles were incrusted. He also noted that plants were stunted and had few leaves in the heavily dusted portions of an alfalfa field downwind from a cement plant in California. Plants appeared normal in another part of the field where there was no visible dust deposit. The dusted plants were also heavily infested with aphis and it was not clear whether the poor growth was due to the aphis feeding or a direct effect of the dust. Entomologists suggested that the primary effect of the dust may have been to eliminate aphid predators, thus encouraging high aphid populations, which in turn cause poor plant growth because of their feeding.

Anderson[9] observed in New York that cherry fruit set was reduced on the side of the tree nearest a cement plant. He demonstrated that the dust on the stigma prevented pollen germination. Schönbeck[10] treated a field of sugar beets biweekly with 2,500 mg/m^2 of dust and observed that infection by leaf-spotting fungus, Cercospora beticola, was significally greater than in nondusted plots. He postulated that the physiological balance was altered by dust increasing susceptibility to infection.

Pajenkamp[11] reviewed the unpublished work of several German investigators, some of whom had applied dust artificially to test plants, and stated that he was opposed to the view that dusts are harmful to plants. He concluded that depositions of from 750 to 1,500 mg/m^2-day (the latter amount representing the maximum that might be found in the vicinity of a cement factory) had no harmful effect on plants.

Raymond and Nussbaum[12] also stated that cement dusts have little effect on wild plants. In papers published with Guderian[13] and Wentzel[14], Pajenkamp stated that limited evidence presented, at best, a contradictory picture, and he acknowledged that the earlier paper[11] had not cited Czaja's work.[5,8,15,16] They also pointed out that a deposit of 1,500 mg/m^2-day was not maximum, since other workers had found up to 2,500 mg/m^2-day. Bohne[6] has since reported weekly averages of up to 3,800 mg/m^2-day.

According to Czaja[5], Ewert concluded that cement-kiln dust did not clog the stomata and that the crust might have a beneficial effect as protection against transpirational losses and a defense against fungi. Czaja also noted that the interpretation of evidence by Ewert is open to question because control plants were heavily infested with flea beetles, while test plants were not.

(3) Dust Components Involved. Detail on the injury to be expected from certain cement-kiln precipitator dusts was given by Czaja.[8] His work is based on comparisons of chemical composition of dusts and resultant injury to leaf cells of a sensitive moss plant, Mnium punctatum. A cut

leaf was mounted in water on a microscope slide, and dust was placed in contact with the water at the edge of the cover slip. Any effect of the resultant solution of leaf cells could be observed directly. Results of tests conducted with 18 dusts fell into the following categories:

a. No permanent injury to living cells, but some plasmolysis;

b. Slight injury to readily accessible cells, disruption of the cytoplasm, and displacement of chloroplasts; and

c. Severe injury to all cells of the leaflet.

Dusts were described as follows: group a, pH of 9.5-11.5, relatively high rates of carbonation (conversion of calcium hydroxide to calcium carbonate), and intermediate amounts (19-29%) of clinker phase (calcium silicates), and characterized by high (36-79%) amounts of secondary salts; group b, pH about 11, high rates of carbonation, lower (13-16%) clinker phase, and characterized by high (81-85%) proportions of raw feed; group c, pH 11-12, very slow carbonation rates, and characterized by high (17-49%) clinker phase. The greater injury was thus related to the larger amounts of clinker phase, which in turn resulted in higher and prolonged alkalinity. But Czaja also pointed out that the composition of dusts within the three groups was not consistent, and that, although not yet demonstrated, the constituents of a given dust undoubtedly influence one another.

In short-term experiments of 2 to 3 days, Darley[3] dusted the primary leaves of bean plants with fractionated precipitator dust obtained from Germany. The dust contained relatively high amounts of potassium chloride, KCl. When a fine mist was applied to dusted leaves, a portion of the leaf tissue was killed (up to 29%), and it was presumed that the action was due to KCl. In later experiments[4] other fractions of the same dust containing very little KCl caused an almost equivalent amount of injury, thus indicating that KCl was apparently not the only factor involved. Current laboratory investigations with different particle-size fractions of precipitator dusts collected around the United States have demonstrated varying degrees of injury to bean leaves when dew is formed on the leaves. There appears to be no effect from dry dusts alone. Inasmuch as these dusts contain very little clinker phase, it is apparent that some components other than those connected with hydration of calcium silicates may also be responsible for injury.

b. Indirect Effects

Pajenkamp[11] reported on unpublished work by Scheffer et al.[17] in Germany during two growing seasons, indicating that even considerable quantities of precipitator dust applied to the soil surface brought about no harmful effects and no other lasting effects on growth or crop yield of oats, rye grass, red clover, and turnips. The dust had a content of about 29.3 percent limestone (analyzed as lime, CaO) and 3.1 percent potassium oxide. The maximum rate of deposit was 0.15 mg/cm^2-day. Discontinuous dustings were made at 0.25 mg/cm^2-day to give an average of 0.075 mg/cm^2-day. In one year, the yield of red clover and the weight of turnips were higher in the dusted plots, although the yield of leaves in the latter crop was reduced. Acid manuring of the soil appeared to increase yield but the interaction of dusting and manuring was not understood.

While Scheffer et al.[17] found no direct injury to plants, they indicated that there might be indirect effects through changes in soil reaction, which in time might impair yield.

Stratmann and van Haut[18] dusted plants with quantities of dust ranging from 0.1 to 4.8 mg/cm^2-day; dust falling on the soil caused a shift in pH to the alkaline side, which was unfavorable to oats but favorable to pasture grass.

2. Fluorides

Particles containing fluoride appear to be much less injurious than gaseous fluorides to vegetation. Pack et al.[19] reported that 15 percent of gladiolus leaf was killed when plants were exposed 4 weeks to 0.79 $\mu g/m^3$ fluoride as HF, but no necrosis developed when plants were exposed to fluoride aerosol averaging 1.9 $\mu g/m^3$ fluoride. Inasmuch as the material was collected from a gas stream which was treated with limestone and hydrated lime, the aerosol was probably calcium fluoride. Moreover, when the accumulated levels of fluoride in leaf tissues were about the same, whether from gas or particles, injury from the latter was much less.

McCune et al.[20] reported an increase of only 4-millimeter tipburn on gladiolus exposed to cryolite (sodium aluminum fluoride dust), wherein the washed leaf tissue from this treatment showed an accumulation of 29 μg/g (29 ppm) fluoride. A 70-millimeter increase in tipburn would have been expected if a similar accumulation had occurred from exposure to HF. Except for the slight tipburn noted above, these authors found that cryolite produced no visible effects on a variety of plants nor did it reduce growth or yield.

It is evident from the work of McCune et al.[20] that fluoride in plant tissue is accumulated from cryolite treatment, but the rate of accumulation is much slower than would be expected from a comparable treatment with HF. For example, when comparing washed leaf samples, exposure of gladiolus to HF for 3 days at 1.01 $\mu g/m^3$ fluoride resulted in an accumulation of 26.4 μg/g (26.4 ppm) fluoride, whereas only 34 μg/g (34 ppm) was accumulated from an exposure to cryolite for 40 days at

1.7 μg/m^3 fluoride. Pack et al.[19] reported only one-third as much fluoride accumulated from particulate matter as from gaseous forms.

Both the investigations cited above indicate that much of the particulate matter remains on the surface of the leaf and can be washed off, although that which remains after washing is not necessarily internal fluoride. Reduced phytotoxicity of particulate fluoride is ascribed in part to the inability of the material to penetrate the leaf tissue. In addition, McCune et al.[20] suggest that inactivity of particles may be due to their inability to penetrate the leaf in a physiologically active form.

3. Soot

Jennings[21] noted the suggestion that soot may clog stomata and prevent normal gas exchange but that most investigations tend to discount this effect. Microscopic examination failed to show enough clogging of stomata on leaves of shade trees (broad-leaved species) to be significant. He further states that interference with light can be more serious but he offers no data from critical experiments to substantiate this theory.

A well-illustrated report by Berge[22] showed plugged stomata on conifers growing near Cologne, Germany. He also stated tht growth was adversely affected.

Necrotic spotting was observed on leaves of several plants where soot from a nearby smokestack had entered a greenhouse.[23] The necrosis was attributed to acidity of the soot particles. Plants outside the greenhouse were not damaged, possibly because the particles had been removed by rain before severe injury could occur.

4. Magnesium Oxide

The possible indirect effect on vegetation of magnesium oxide falling on agricultural soils was reported by Sievers.[24] He noted poor growth in the vicinity of a magnesite-processing plant in Washington. Experiments were designed to grow plants in soil collected at various distances from the processing plant, in normal soil and in soil to which magnesium oxide was added. Suppression of plant growth was demonstrated with the high levels of magnesium. After the processing plant ceased operation, injury to crops in the area became less pronounced indicating that the injurious effect was not a permanent one.

5. Iron Oxide

Berge,[25] in Germany, dusted experimental plots with iron oxide at the rate of 0.15 mg/cm^2-day over 1- to 10-day intervals through the growing season for 6 years. The plots were planted with cereal grains or turnips, and effects of treatment on the primary product, on straw, and on leaves were noted. No harmful effect of the dust was detected on either crop. There was a tendency for improvement of yields of grain and turnip roots, but this was not statistically significant.

6. Foundry Dusts

Changes in starch reserves were compared in common holly leaves, untreated and treated with dusts emitted from foundry operations.[6] The critical factor was the amount of light absorbed by the dust layer, and the influence on transpiration or overheating of leaf tissue was of minor significance. These observations agree with some of those reported above on the range of effects of cement-kiln dust on vegetation.

7. Sulfuric Acid Aerosols

These particles too may settle on plants and cause injury. They are not discussed here, however, as the subject is covered in some detail in a companion document, Air Quality Criteria for Sulfur Oxides.

8. Lignite and Coal Fly Ash

Holte[26] and Kick and Grosse-Brauckmann[27] analyzed both coal and lignite fly ash and reported that phytophysiologically the following components are readily avilable: CaO, 42 percent; MgO, 7.2 percent; and SO_3, 10 percent. They also detected considerable amounts of aluminum and iron, but the effects of these on plants should be small.

According to Knickmann[28] and Monning[29], coal fly ash has a markedly smaller content of calcium, magnesium, and sulfur. They found that fly ash from thermal power plants will reduce the soil acidity. Consequently, the effects noted by V. Bombard[30] might be attributed to change in the alkalinity of the soil by fly ash. In sandy soil (pH 6.8 to 7.0), the yields of rye and potatoes are reduced by the addition of fly ash.

C. EFFECTS ON ANIMALS OF DUSTS INGESTED WITH VEGETATION

Particles which contain chemical components detrimental to animal health may be assimilated through ingestion of plant materials. The toxic components may be absorbed into the plant tissues or may remain as a surface contaminant on the plants. When evaluating the potential harm to animals from ingested vegetation, both absorbed and deposited particles should be considered.

Fluorosis in animals has been reported from ingestion of vegetation covered with a fluoride-containing particulate matter.[31] Injury occurs when absorbed plus deposited fluoride on the plants reached 40 to 50 μg/g. Arsenic poisoning of cattle and sheep has occurred from ingestion of arsenic-containing particles settled on vegetation.[32]

D. SUMMARY

There has been relatively little research on the effects of particulate matter on vegetation, and most of the experiments done to date have dealt with specific kinds of dusts rather than the conglomerate mixture normally encountered in the atmosphere.

The significance of dusts as phytotoxicants is not yet clear, but there is considerable evidence that certain fractions of cement-kiln dusts adversely affect plants when naturally deposited on moist leaf surfaces. Dry cement-kiln dusts appear to have little deleterious effect, but in the presence of moisture the dust solidifies into a hard adherent crust which can damage plant tissue and inhibit growth. Moderate damage has been observed on the leaves of bean plants dusted at the rate of about 0.47 mg/cm^2-day (400 $tons/mi^2$-mo) for 2 days and followed by exposure to naturally occurring dew. Similarly, a marked reduction in the growth of poplar trees 1.6 km (1 mile) from a cement plant was observed after cement production was more than doubled. At levels in excess of 0.1 mg/cm^2-day (85 $tons/mi^2$-mo), incrustations have been observed on the branches of fir trees, with the result that needles fell prematurely, shortening the succeeding year's flush of growth. Although the mechanism by which injury occurs is not entirely understood, it is possible that the crust intercepts the light needed for photosynthesis and starch formation, causes alkaline damage to tissues, and prevents normal gas exchange in leaf tissues. Injury due to the direct effect of high pH on cell constituents does occur. Plugging of stomata and reduced growth of trees may occur within a short distance of cement plants.

It should be noted, however, that the harmful effect of cement dusts on vegetation is not fully substantiated and has been questioned by some workers. The controversy that surrounds this subject is not surprising in view of the limited research to date. In addition, not all studies have been carried out under identical conditions or with dusts of the same composition. Studies of the effects of cement-kiln dusts deposited on the soil also raise questions. Some investigators report no harmful effects at levels from 750 to 1,500 mg/m^2-day (130 to 640 tons/mi^2-mo), while others report that concentrations from 0.1 to 4.8 mg/cm^2-day (86 to 4,000 tons/mi^2-mo) cause shifts in the soil alkalinity which may be favorable to one crop but harmful to another.

Fluorides in particulate form are less damaging to vegetation than gaseous fluorides. Fluoride may be absorbed from depositions of soluble fluoride on leaf surfaces. However, the amount absorbed is small in relation to that entering the plant in gaseous form. The fluoride from particles apparently has great difficulty penetrating the leaf tissue in a physiologically active form. The research evidence suggests that few if any effects occur on vegetation at fluoride particle concentrations below about 2 μg/m^3. Concentrations of this magnitude can be found in the immediate vicinity of sources of fluoride particulate pollution, but they are rarely found in urban atmospheres. Fluorides absorbed or deposited on plants may be detrimental to animal health. Fluorosis in animals due to the ingestion of vegetation covered with particles containing fluorides has been reported. In a similar manner arsenic poisoning of cattle and sheep has occurred from ingestion of arsenic-containing particles that had settled on vegetation.

Soot may clot stomata and may produce necrotic spotting if it carries with it a soluble toxicant, such as one with excess acidity. Magnesium oxide deposits on soils have been shown to reduce plant growth, while iron oxide deposits appear to have no harmful effects and may be beneficial. Sulfuric acid aerosols will cause leaf spotting. The levels at which these materials may produce a toxic response are not well defined.

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Chapter 7

SOCIAL AWARENESS OF PARTICULATE POLLUTION

A. INTRODUCTION

The nuisance of air pollution is to some extent a subjective perception, and its significance is therefore influenced to some extent by the way the public feels about pollution. Nevertheless, it is acceptable practice to conclude that nuisance and levels of pollution are related if it can be demonstrated that a nuisance response by a sample population is positively related to average levels of actual pollution. The development of such evidence is difficult because public reaction probably reflects pollution peaks rather than the mean value for an extended time period. This problem has been considered by McKee.[1]

Several studies have attempted to assess the annoyance to a population of community air pollution, but not all have taken advantage of available aerometric data to establish the relationships between air pollution levels and attitudes and opinions among the affected population. The chief value of these surveys lies in demonstrating that a significant proportion of the public is aware of, and concerned about, air pollution and is willing to pay to abate the nuisance.[2-4] One of the most obvious indications of the nuisance of air pollution is citizen complaints. Generally speaking, the person who is willing to take the time to telephone or write a complaint about air pollution is probably seriously irritated by it. Those who actually complain may represent only a small proportion of the total number of persons who find air pollution objectionable. There are many who may be irritated who will not complain because they do not believe complaining will do any good. There are others, conspicuous by their absence, who have moved away because of the effects of air pollution on their health or because of the aesthetic degradations of their neighborhood.[5] Unfortunately, very little work has been done in this particular area of measuring the response of the public to the nuisance of air pollution.

It is important to note that application of present control technology may result in a much larger reduction in larger particles than in smaller particles. The remaining smaller particles will not be as readily discernible to the public, and awareness of particulate pollution may diminish.

The effect of particulates on the total ecological system and man's enjoyment of this environment cannot as yet be fully evaluated.

B. THE STUDIES

1. St. Louis, Missouri

One major investigation of the relationship between public opinion and particulate air pollution concentrations is that conducted in the Greater St. Louis area.[3,6] This area included most of the population residing in the St. Louis, Missouri, metropolitan area. Persons interviewed were asked whether they believed that air pollution was present and whether they regarded it as a nuisance. The responses were related to the pollution level measured both as suspended particle concentration and as soiling index. See Chapter 1 for a discussion of this latter measure of pollutant concentration.) Figure 7-1 shows the results obtained for both questions in terms of suspended particle concentrations.[6] It will be seen that the population becomes aware of pollution before it regards it as a nuisance. Where the average annual geometric mean concentration of particles was 80 $\mu g/m^3$, better than 30 percent of the population indicated awareness of air pollution. Fifty and 75 percent of the survey population were aware of air pollution in the area where the average annual levels of particulate matter were 120 and 160 $\mu g/m^3$ respectively. None of the population appeared to be bothered by particle concentrations below 50 $\mu g/m^3$, 10 percent of the population was bothered at a level of about 80 $\mu g/m^3$, 20 percent was bothered about 120 $\mu g/m^3$, 33 percent at 160 $\mu g/m^3$, while 40 percent regarded a concentration of 200 $\mu g/m^3$ as constituting a nuisance.

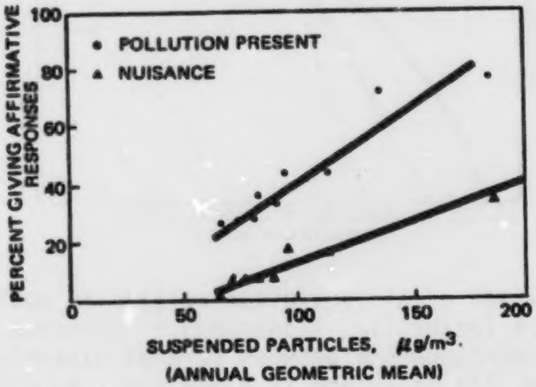

Figure 7-1. Proportion of population in St. Louis stating air pollution present in their area of residence, and proportion of St. Louis population bothered.[6] (This figure presents the result obtained from interviews.)

2. Nashville, Tennessee

A study by Smith et al.[7] reveals the complexity of public opinion surveys. The method used was similar to that in other surveys: public opinion data were compared with aerometric data from the nearest air sampling station. One group of questions in the public opinion survey dealt specifically with nuisance aspects of air pollution. The proportion of affirmative responses to the question concerning too much dust and dirt on porch and window sills showed a clear increase with increasing particle concentrations. The data do not, however, permit a predictable quantitative statement of this responsibility.

Survey results indicated that up to 3.8 percent of the respondents voluntarily expressed awareness and concern about air pollution as a health problem. In response to a direct question, 23 percent of the

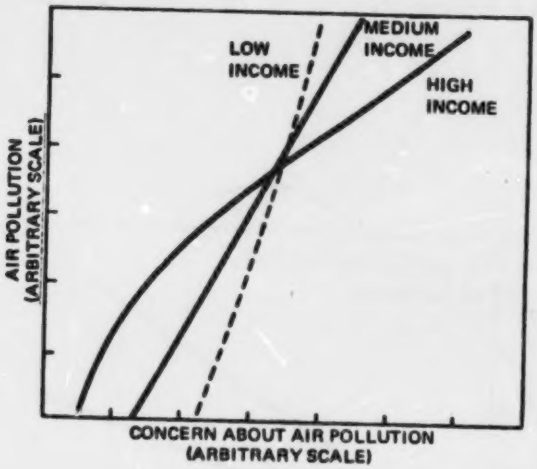

Figure 7-2. The level of air pollution related to public opinion in three income groups (Nashville, Tennessee).[7] (Figures 7-2 and 7-3 show the interlocking relationships between several parameters affecting public concern over air pollution in Nashville. At high levels of pollution, concern is strongest in those of highest socioeconomic status, while at low pollution levels, concern is greatest among those of low status. This result is possibly connceted with the radial distance from Nashville at which persons of different socioeconomic status reside.)

respondents stated they were bothered in some way by air pollution. The data indicate that the frequency of occurrence of days of acute pollution had a direct influence on the proportion of people who would express concern and annoyance.

The study indicated some relationships between level of concern and socioeconomic status. At high levels of air pollution, concern was greater among women of high socioeconomic status than among women of low socioeconomic status; although at low levels of air pollution, those of low socioeconomic status expressed more concern than those of high status. Figures 7-2 and 7-3 together demonstrate

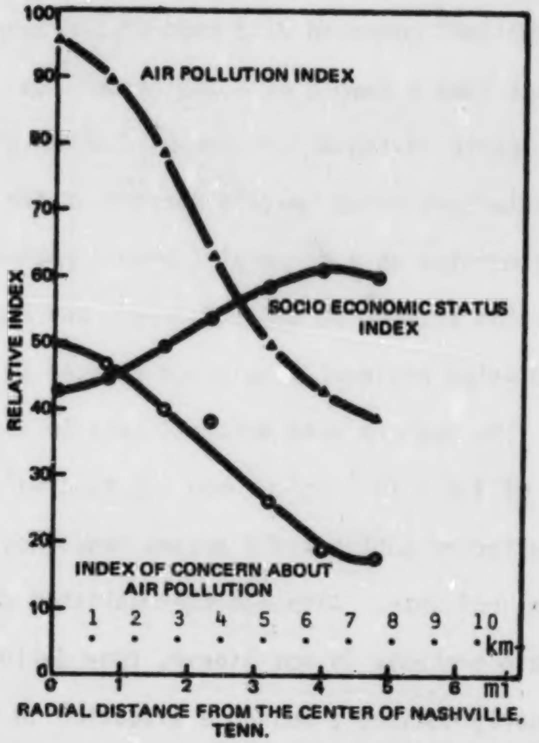

Figure 7-3. Radial distribution from the center of Nashville of air pollution levels, socioeconomic status, and public concern about air pollution.[7]

the interlocking relationship between socioeconomic status, distance of residence from the center of the city, and concern about air pollution.[7] Figure 7-2 uses arbitrary scales for both variables and should not be taken to indicate that exact form of the relationships.

Also, using Nashville as a survey area, Williams and Edmisten[8] found acceptable the hypothesis that perception of and for air pollution is directly related to neighborhood pollution levels. Their study also showed that the citizen's perception of air pollution was influenced more by the frequency of occurrence of high daily concen-- trations of particles than by high monthly, seasonal, or annual average concentrations.

3. Birmingham, Alabama

Stalker and Robison[9] compared data from 21 air sampling stations with public responses from a sample of 7,200 households. Data were gathered only from people living within one mile of an air sampling station. The investigators found that 10 percent of the population believed a nuisance existed at a seasonal (summer) mean suspended particle Aconcentration between 60 and 180 $\mu g/m^3$, and more than 30 percent of the population believed a nuisance existed at levels between 100 and 220 $\mu g/m^3$. The authors used dustfall data to demonstrate that for each increment of 3.5×10^{-1} mg/cm^2-mon (10 $tons/mi^2$-mo) another 10 percent of the affected public would become concerned and consider that level a general nuisance. Although the published data indicate that the relationship probably is not linear, they do indicate that the respondents clearly defined a nuisance situation in their area of residence at annual mean dustfall values of 3.5×10^{-1} mg/cm^2-mo or above.

4. Buffalo, New York

De Groot and Samuels[2] used two independent samples taken three years apart to determine public opinion. Opinions were assessed against aerometric data from the nearest of nine air sampling stations. The measure of public opinion was the percentage of the population considering air pollution a serious community problem. The investigators counted only clear, unequivocal responses; respondents who indicated uncertainty about the seriousness of the problem were assigned to the "not serious" category. In Buffalo, as in Birmingham, there were apparent positive relationships between public awareness and levels both of dustfall and of suspended particles. There was also a positive relationship between the frequency of days of acute levels of these pollutants and public opinion.

C. SUMMARY

Public opinion survey data in several cities indicate a positive relationship between the concern expressed about air pollution by a population and the actual levels of particulate pollution--used as an index of air pollution. In general, over the ranges studied, an increasing proportion of the population expresses dissatisfaction over air pollution as concentrations of particulate matter increase. However, while the several studies agree, for the most part, in this qualitative relationship, it is difficult to compare the studies qualitatively. Each study used different sampling schemes, questionnaire schedules, and methods of interviewing. In addition, the socioeconomic characteristics of the population sampled varied from city to city.

The St. Louis study showed that 10 percent of the study population was bothered by air pollution in areas where the annual geometric mean value of suspended particulates was about 80 $\mu g/m^3$. About 20 percent of the study population was bothered in areas with an annual geometric mean value of 120 $\mu g/m^3$ and 33 percent with a mean of 160 $\mu g/m^3$. The responses are probably affected by the short-term fluctuations in particulate levels which are associated with any time-averaged data.

Other studies show that when dustfall levels exceeded an annual mean of 3.5×10^{-1} mg/cm^2-mo (10 $tons/mi^2$-mo, at least 10 percent of the affected population expressed concern about a nuisance situation.

The available literature indicates that the extent to which a population considers air pollution an annoyance is related to the frequency of occurrence of days with acute pollution as well as to the average level, that the level of concern is related to socioeconomic status, and that the population becomes aware of pollution at particle concentrations lower than those at which they consider that it constitutes a nuisance.

In the St. Louis study, 30 percent of the study population was aware of pollution in areas where the annual geometric mean concentrations of suspended particulates was 80 $\mu g/m^3$, 50 percent in areas with 120 $\mu g/m^3$ and 75 percent in areas with 160 $\mu g/m^3$.

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Chapter 8

ODORS ASSOCIATED WITH ATMOSPHERIC PARTICULATE MATTER

A. INTRODUCTION

The human olfactory sense has the ability to detect and respond to thousands of different materials or chemical compounds. Some odorants can be detected in concentrations as low as 1 ppb. Available instrumentation lacks both the selectivity and the sensitivity possessed by the olfactory sense for the detection of many odorants.

Odorants in themselves do not cause organic disease; however, do discomfort and disagreeableness brought about by obnoxious odors can cause some temporary ill effects. The effects that border upon ill health include lowered appetite, lowered water consumption, impaired respiration, nausea, vomiting, and insomnia.

Particles themselves are not considered capable of directly stimulating the olfactory sense. However, this should not be interpreted to mean that all airborne particulate matter, as a pollutant category, is incapable of stimulating the odor sense nor that particles cannot be involved in the delivery of the odorant to the receptor cells. There is evidence that some particles can stimulate the sense of smell because the particle itself is volatile or because this particle is desorbing a volatile odorant.[1] There is also speculation that some particulate matter is capable of stimulating the sense of smell.[2] Regardless of the mechanism of how particles are involved in the stimulation of the olfactory sense, the important fact is that they definitely appear to be involved.

B. EVIDENCE FOR THE ASSOCIATION OF ODOR WITH PARTICLES

The idea that odors may be associated with particles is supported by observations that filtration of particles from an odorous airstream can reduce the odor level. Rossano and Ott[2] showed that the removal of particulate matter from diesel exhaust by thermal precipitation effected a marked reduction in odor intensity. The precipitation method was selected because it provides minimal contact between the collected particles and the gaseous components of the diesel exhaust stream. Thus, effects that could be produced by a filter bed, such as removal of odorous gases by adsorption in the filter cake, are eliminated. The observed odor reduction must, therefore, have resulted directly from the removal of particulate matter. The particles collected by Rossano and Ott were aggregates of spherical balls about 0.04 to 0.05 μm in diameter. Particulate matter from diesel exhaust collected on glass fiber filters by Linnell and Scott[3] yielded a heavy "diesel" odor. Analysis of the filtrate showed that it contained no acrolein or formaldehyde, but it did release NO_2 when it was heated to 100° C. The authors conclude that "no appreciable gas phase concentration changes for acrolein or formaldehyde will result from particulate removal." Other more or less casual observations on the role of particulate matter in community odor nuisance problems appear occasionally in the literature.[4]

A second piece of evidence that implicates the association of particles with odors is the production of a mild odor, sometimes described as "yeasty," in air that is passed through a bed of activated carbon. Turk and Bownes[5] have reported that this odor is not produced by any detectable desorption of gaseous matter from the carbon and that the same odor can be produced by passing air through silica gel.[6] It is possible that subfilterable particles are associated with this phenomenon.

C. HYPOTHETICAL MECHANISMS FOR THE ASSOCIATION OF ODOR WITH PARTICLES

1. Volatile Particles

Liquid or even solid aerosols may be sufficiently volatile that their vaporization on entering the nasal cavity produces enough gaseous material to be detected by smell. Such aerosols may be relatively pure substances, such as particles of camphor, or they may be mixtures which release volatile components. The retention of the odorous properties by volatile aerosols will, of course, depend on their vapor pressures and their ages.

2. Desorption of Odorous Matter by Particles

Goetz[7] has treated the kinetics of the interaction between gas molecules and the surface of airborne particles. His theoretical considerations were directed to the question of transfer of toxicants by particles, but are also applicable to odors. Even if a given aerosol is intrinsically odorless, it could act as an odor intensifier if (1) the sorptive capacity of the aerosol particles for the odorant smaller than the affinity of the odorant for the nasal receptor, and at the same time (2) the sorptive capacity of the aerosol particles is large enough to produce an accumulation of odorant on the particle surface. Such aerosol particles would concentrate odorous molecules on their surfaces, but the odorous matter would be transferred to olfactory receptors when the aerosol entered the nasal cavity. The odorous matter would then be present at the receptor sites in concentrations higher than in the absence of the aerosol. The resulting effect would be synergistic. If an odorant is more strongly adsorbed by the aerosol particles than by the olfactory receptors, transfer of the odorant to the receptors would be impeded and the particles would actually attenuate the odor.

3. Odorous Particles

No study has rigorously determined the upper limit of particle size for airborne odorous matter. Particles up to about 8 or 10 x 10^{-4} μm in diameter are considered to be molecules that can exist in equilibrium with a solid or liquid phase from which they escape by vaporation. The vapor pressure decreases as the molecular weight increases, and particles above about 10^{-3} μm do not generally exist in any significant concentration in equilibrium with a bulk phase; hence we do not consider them to be "vapors." Nonetheless, it is possible that odorant properties do not disappear when particle sizes exceed those of vapor molecules. Knowledge about particles in the size range of 1 to 5 x 10^{-3} μm (up to about the size of small viruses) is relatively meager, and whether or not they can be odorous, or what the effect of an electrical charge on their odorous properties might be is not known. Larger particles may also be intrinsically odorous, although their more significant role may be to contribute to odor transport by absorbing and desorbing odorous gases and vapors.

D. COMMON ODOR PROBLEMS

Kerka and Kaiser[8] surveyed State and local air pollution control personnel in the United States and compiled the list of most frequently reported odors shown in Table 8-1.

Of the 35 listings in Table 8-1, nine are either known or suspected to be particulate-borne. These nine include gasoline- and diesel-engine exhaust, coffee roasting, restaurant odors, paint spraying, roofing and street paving, asphalt manufacturing, home

Table 8-1. MOST FREQUENTLY REPORTED ODORS[8]

Source of odor	*Number reported*
Animal odors:	
Meat packing and rendering plants	12
Fish oil odors from manufacturing plants	5
Poultry ranches and processing	4
Odors from combustion processes:	
Gasoline and diesel engine exhaust	10
Coke-oven and coal-gas odors (steel mills)	8
Maladjusted heating systems	3
Odors from food processes:	
Coffee roasting	8
Restaurant odors	4
Bakeries	3
Paint and related industries:	
Manufacturing of paint, lacquer, and varnish	8
Paint spraying	4
Commercial solvents	3
General chemical odors	
Hydrogen Sulfide	7
Sulfur Dioxide	4
Ammonia	3
General industrial odors	
Burning rubber from smelting and debonding	5
Odors from dry-cleaning shops	5
Fertilizer plants	4
Asphalt odors—roofing and street paving	4
Asphalt odors—manufacturing	3
Plastic manufacturing	3
Foundry odors	
Core-oven odors	4
Heat treating, oil quenching, and pickling	3
Smelting	2
Odors from combustible waste:	
Home incinerators and backyard trash fires	4
City incinerators burning garbage	3
Open-dump fires	2
Refinery odors:	
Mercaptans	3
Crude oil and gasoline odors	3
Sulfur	1
Odors from decomposition of waste:	
Putrefaction and oxidation—organic acids[a]	3
Organic nitrogen compounds—decomposition of protein[a]	2
Decomposition of lignite (plant cells)	1
Sewage odors:	
City sewers carrying industrial waste	3
Sewage treatment plants	2

[a] Probably related to meat processing plants.

incinerators, and backyard trash fires, city incinerators burning garbage, and open-dump fires.

E. SUMMARY

Airborne particulate matter, as an air pollutant category, is normally not considered as a source of odor stimulation. However, there is evidence that some particulates having volatile components can produce an odor response in human receptors.

Further, by a suggested mechanism of adsorption and subsequent desorption, an odorant may be transferred by a particulate substrate. When one examines the various types of odor sources which result in public awareness of an undesirable situation, a significant number of these are either known or suspected to be particulate-borne. The sources of these odorous particles are diverse, including diesel and gasoline engine exhausts, coffee-roasting operations, paint spraying, street paving, and the burning of garbage and trash. Despite the absence of an exact mechanism to explain the association of odor with particulates, intimate involvement in multiple categories of citizen nuisance complaints cannot be ignored.

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Chapter 9

THE RESPIRATORY SYSTEM: DEPOSITION, RETENTION, AND CLEARANCE OF PARTICULATE MATTER

A. INTRODUCTION

In urban communities, exposure to atmospheric pollutants may constitute a health hazard that is the most serious single consequence of air pollution. Chapters 10 and 11 discuss the effects of particulate pollutants on health in terms of toxicological and epidemiological studies. Pollutants are likely to enter the human body mainly via the respiratory system; other routes of entry are of minor importance. Damage to the respiratory organs may follow directly, or the pollutant may be transported by some mechanism to a remote susceptible organ. It is apparent that a study of the effect on health of particulate atmospheric pollutants requires an understanding of the mechanisms and efficiencies of deposition of particles in the respiratory system, and of the subsequent retention within, and of clearance from the system, as well as the secondary relocation of pollutants to other sites in the body. This chapter provides a brief introduction to the physics and physiology of deposition, retention, and clearance in the respiratory system. More complete descriptions may be found in several reviews.[1-3]

Experimental studies of the several factors involved in deposition, retention, and clearance processes have been backed up by theoretical treatments and, in the discussion which follows, descriptions of these theoretical models precede those of experimental work. One of the latest theoretical models is that developed by the Task Group on Lung Dynamics for Committee II of the International Commission on Radiological Protection.[4] The Task Group's report establishes the usefulness of the aerodynamic (mass median) diameter (cf. Chapter 1-A) of a particle as a measure of deposition probability. This is of practical importance, since the aerodynamic diameter may readily be determined under field conditions.

The anatomy of the respiratory system plays an important part in determining the effects of inhaled particles, and a very short description of this anatomy is provided. Several extensive presentations of morphological studies are available,[5,6] and Davies[7] gives a formalized concept of the anatomy of the human respiratory tract.

B. ANATOMY OF THE HUMAN RESPIRATORY TRACT

The respiratory system, for convenience, is considered to consist of three main sections:

1. the nasopharyngeal structure,
2. the tracheobronchial system, and
3. the pulmonary structure within which oxygen and carbon dioxide are exchanged between respired air and blood.

Figure 9-1 shows the location of these features. The nasal passages lead, via the nasopharyngeal structure and the larnyx, to the trachea and the bronchi, which are made up of 23 generations of dichotomous branching tubes terminating in the alveolar (air) sacs. Figure 9-2 is a schematic representation in greater detail of the terminal bronchioles and the pulmonary structure.

Estimates of numbers of alveoli differ somewhat from one experimenter to another: a recent suggestion[8] is that there are 300 million, together with 14 million alveolar ducts. The alveoli are probably between 150 and 400 μm in diameter, so that the total alveolar surface varies between about 30 and 80 m^2. The increasing total

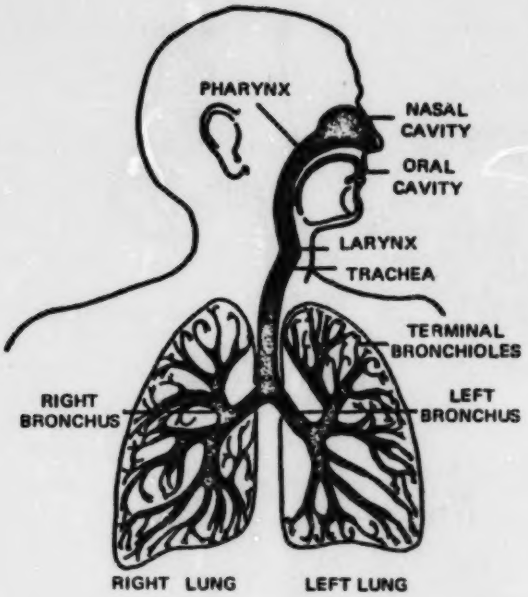
PHARYNX
NASAL CAVITY
ORAL CAVITY
LARYNX
TRACHEA
TERMINAL BRONCHIOLES
RIGHT BRONCHUS
LEFT BRONCHUS
RIGHT LUNG
LEFT LUNG

Figure 9-1. The major anatomical features of the human respiratory system. (The diagram shows the major divisions of the human respiratory tract into nasopharyngeal, tracheobronchial, and pulmonary compartments.)

cross-sectional area with progression down the respiratory tract leads to a marked decrease in the velocity of air movement with depth.

The nasopharyngeal and tracheobronchial structures possess ciliated epithelium covered with mucus arising from goblet cells and secretory glands. These structures also make up the anatomical dead space, since oxygen exchange between the blood and air does not occur here. The surface of the pulmonary structure consists of nonciliated moist epithelium that, although devoid of the secretory element found in the tracheobronchial tree, is covered by a surface-active material, without which alveoli would totally collapse (atelectasis) at the end of respiration.

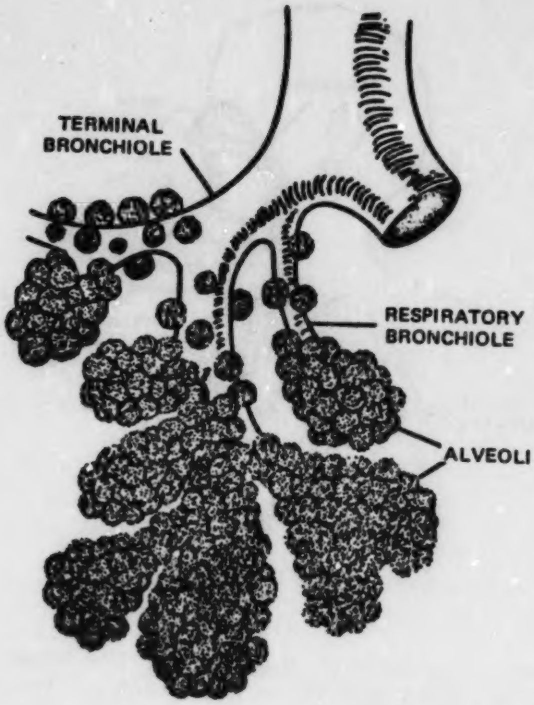

Figure 9-2. The terminal bronchial and alveolar structure of the human lung. (The diagram shows the pulmonary structure of the respiratory tract.)

C. FACTORS AFFECTING THE DEPOSITION AND RETENTION OF PARTICULATE MATTER IN THE RESPIRATORY SYSTEM

1. Physical-Mathematical Treatments

The theoretical physical-mathematical treatments use information on the anatomical structure and airflow rates in the respiratory tract, together with knowledge of the physical factors influencing the deposition of particles. The deposition mechanisms will be considered first; aerodynamic factors connected with airflow and gas mixing in the respiratory system will then be mentioned before proceeding to a discussion of the three main models which have been developed.

a. Deposition Mechanisms

Three mechanism are of importance in the deposition of particulate matter in the respiratory tract--inertial impaction, gravitational settling (sedimentation), and diffusion (Brownian motion). The relative significance of these deposition mechanisms varies with anatomical and physiological factors, with the nature of the airflow, and with the characteristics of the aerosol such as particle shape and density.

Particles in an airstream impinge onto a surface when the inertia of the particle is great enough to overcome the resistive forces of the medium. If gravity is neglected, the path of the particle is determined by the air velocity, the mass and air resistance of the particle, and the size and shape of the obstacle. Inertial impaction is therefore of greatest importance in the deposition of large particles of high density and at branching points in the respiratory airway system where the direction of flow changes.

The size of the particle as well as its density is a significant factor in determining the importance of deposition by gravitational settling. The terminal velocities (settling velocities) of spheres of unit density in air for 10-, 1-, and 0.1-μm particles are 3×10^{-1}, 4×10^{-4}, and 8×10^{-5}cm/sec, respectively. Density also plays a part since the sedimentation behavior of a 0.5-μm particle of density 10 g/cm^3 would be equivalent to that of a unit density particle of 1.5-μm diameter. Hence, gravitational settling is most important in the deposition of large particles or of high-density particles such as dusts of heavy metals. Irregularly shaped particles will have an aerodynamic diameter less than the measured geometric diameter.

The third main mechanism active in bringing about deposition of particles in the respiratory tract is Brownian movement or diffusion; it is the result of bombardment of the particles by air molecules which are in rapid, random, thermal motion. Diffusion is negligible for large particles (say above 0.5-μm diameter), while it may be the major mechanism for the deposition of small particles (below 0.1 μm) in the lower pulmonary tract, where airflow rates are lower and the distances to the walls are less.

b. Aerodynamic Factors

Two aerodynamic factors, namely, flow rates and gas mixing, are incorporated in the models of the respiratory system.

During the respiratory cycle, the actual airflow rate varies from zero up to a maximum value and then back down to zero. Usually the expiratory phase is longer than the inspiratory phase and there may be pauses between the two phases. A study of the respiratory airflow patterns of healthy young men at rest and under a wide range of work loads was made by Silverman et al.[9] The maximum inspiratory flow rate increased from a mean value of 40 l/min in sedentary subjects to 100 l/min at an exercise level of 622 kg-m/min and to 286 l/min at an exercise level of 1,660 kg-m/min. The corresponding values for maximum expiratory flow rates were 32, 107, and 322 l/min. The collected results are shown in Table 9-1.

Changes in flow rate resulting from physical activity have a profound effect on particle deposition in the respiratory system; this effect in turn depends upon the aerodynamic size of the particulate material inhaled. It has been demonstrated by Amdur, Silverman, and

Table 9-1. RESPIRATORY AIRFLOW PATTERNS FOR A GROUP OF HEALTHY YOUNG MEN.[9]

Exercise level	Inspiratory flow rate, l/min (max)	Expiratory flow rate, l/min (max)
Sedentary.	40	32
622 kg-m/min	100	107
1660 kf-m/min.	286	322

Drinker[10] that the inhalation of irritant particulate material, such as sulfuric acid mist, can decrease the maximum inspiratory and expiratory flow rate in human subjects. Such alterations produced by irritant particles could also affect deposition patterns of particulate matter in general, and may represent the physiological defense response of the body.

Another factor to be considered in the deposition of particulate matter in the lung is the role of mixing of intrapulmonary gas flow. A study of this factor has been made by Altshuler and coworkers[11] using 0.4-μm particles suspended in air. From the measured wash-in and washout rates (after return to particle-free air), the authors were able to calculate the volume of new air which mixed with the residual air. Their data showed that at a tidal volume of 500 ml, not more than 11 to 27 percent of new air in each successive breath actually mixed with the residual air. It follows that nondiffusible particles (above 0.5 μm) will tend to penetrate only to the depth of the new air, while smaller particles will have great enough diffusion velocities to move independently into the static air in the lung, in the same way that gas molecules do.

c. The Models

For the present purposes, the most important model is that of the Task Group on Lung Dynamics.[4] However, two earlier studies, those of Findeisen[12] and Landahl,[13] are of significance, since they were both considered as a basis for the Task Group model. The Task Group finally selected Findeisen's anatomical model, since it appeared that the rather more sophisticated treatment of Landahl gave no better estimates of deposition values. Further, the cumulative volume down to the end of the terminal bronchioles in the Findeisen model is more in keeping with the anatomical dead space as determined by physiological measurements.

Findeisen[12] predicted the percentage of deposition and the site of deposition of particles of various sizes in the respiratory tract. He estimated there would be a critical particle size of about 0.3- to 0.4-μm radius for which a minimal amount of 34 percent would be deposited. This deposition would occur predominantly in the terminal airways and alveoli, thus suggesting that particles of this size should not be dismissed as toxicologically unimportant.

Findeisen's concepts of the anatomy of the pulmonary tract have been criticized by Weibel.[14] Findeisen assumed equal and constant flow rates for inspiration and expiration, a situation which does not prevail during the respiratory cycle. Furthermore, this simple respiratory pattern does not take into account the unique distribution of air in the lungs or the factors of intrapulmonary mixing of tidal and residual air in the lungs.

Landahl[13] made a similar theoretical study of the problem of particle deposition in the respiratory tract, but included the mouth and pharynx in his anatomical model. He employed several tidal volumes and breathing frequencies in his calculations, but in all cases assumed a constant flow for both the inspiratory and expiratory phases. The calculations yielded deposition values for both phases and took into account the progressively smaller fraction of each tidal volume which penetrated to the various depths. Deposition in various regions for spheres of unit density in the size range 20 to 0.2 μm was predicted to fall to a minimum at the size where the predominant force bringing about deposition is shifting from gravitational settling to diffusion. An increase was found in retention with larger tidal volumes.

Landahl[15] also made a separate consideration of deposition in the nasal passages. Earlier studies did not take into account impaction of particles on nasal hairs, or deposition by inertial forces as the airflow changes direction, or sedimentation within the nasal chamber. Assuming a flow rate of 18 l/min, about 75 percent of 10-μm particles would be retained in the nose. Corresponding values would be about 50 percent for 5-μm particles and 10 percent for 1-μm particles. Landahl calculated that at flow rates greater than 18 l/min, essentially 100 percent of the particles above 10 μm would be retained in the nose and that there also would be substantail retention of 2- to 5-μm particles. Nasal deposition is negligible for particles below 1μm in diameter. It follows that the estimated pulmonary deposition values of Findeisen would have to be revised downward for the large particles.

The Task Group on Lung Dynamics used the conventional division of the respiratory tract into three compartments and made three fundamental assumptions in the development of their model. These were:

1. The log-normal (Chapter 1-A) frequency distribution is generally applicable to particle sizes in the atmosphere.

(It should be noted that this assumption is by no means universally accepted.)

2. The physical activity of the individual, affects deposition primarily by its action on ventilation, since physiological adjustment to the demands of increased minute ventilation is to increase tidal volume more than respiratory frequency. In terms of producing the greatest change in deposition throughout the respiratory tract, the effect of an increase in volume at a constant respiratory frequency was considered. (A frequency of 15 breaths per minute was used together with tidal volumes of 750, 1,450, and 2,150 ml (BTPS)*; the lowest tidal volume is considered representative of a mild-to-moderate activity state.)
3. The aerodynamic properties of the particle, the physiology of respiration, and the anatomy of the respiratory tract provide a basis for a meaningful and reliable deposition model.

The Task Group's aim was to amalgamate size-deposition relationships into a generalized form which would directly permit the prophetic use of dust sampling information. By conventional sampling methods, the count median diameter or the mass median diameter is obtainable along with the geometric standard deviation, σ_g (Chapter 1-A). The measurement of one diameter permits the calculation of the other, and both can be expressed as aerodynamic equivalent diameters. The Task

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*BTPS indicates volume computed at body temperature and pressure for air saturated with water vapor.

Group used calculations basically similar to those of Findeisen[12] to estimate the deposition in the three respiratory compartments. The results of one set of these calculations are shown in Figure 9-3.[4] For this illustration, a tidal volume of 1,450 ml has been used, and the log-normal dust distribution has been expressed by the aerodynamic mass median diameter and a geometric standard deviation ranging from 1.2 to 4.5. Each shaded envelope indicates the range of the calculated fractions of the inhaled mass of dust of a given size that would be retained in each respiratory compartment. Within the particle size

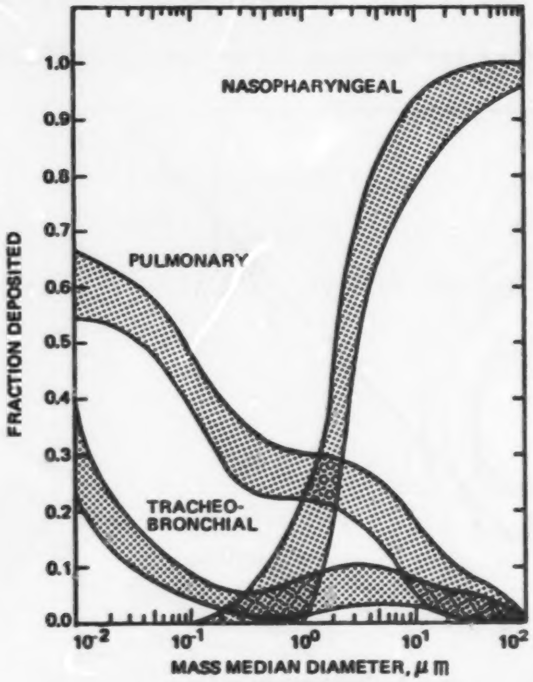

Figure 9-3. Fraction of particles deposited in the three respiratory tract compartments as a function of particle diameter.[4] (This figure shows the deposition efficiencies calculated by the Task Group on Lung Dynamics.)

limits 0.01 to 100 μm, the aerodynamic mass median diameter served to characterize the deposition probabilities of the entire particle size distribution from which it emerged. Other parametric functions of the particle distribution failed to produce such simple cohesive relationships.

The effect of the varying tidal volumes is shown in the original report (Figure 12, reference 4), but it is possible, for practical purposes, to use a mean curve for 1,450 ml to represent all three respiration states. This implies that the minute volume will control the total amount of particulate material deposited but will not ordinarily have much effect on the percentage deposition.

The final step was to plot the combination of mean distribution versus mean respiratory performance curves on log-probability paper, a

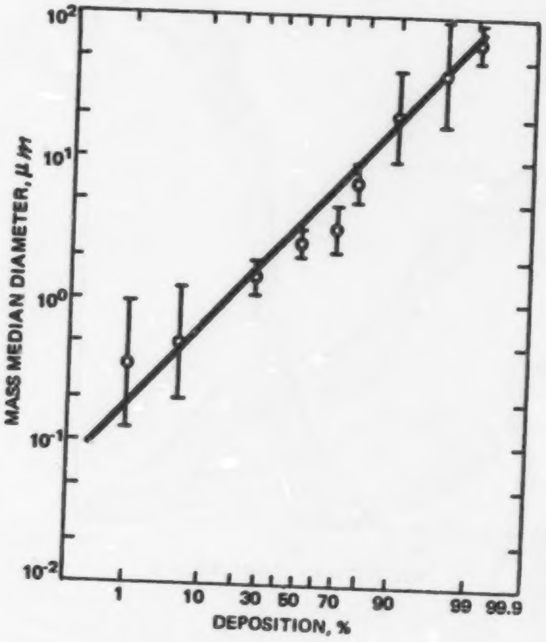

Figure 9-4. Data from Figure 9-3 for the nasopharyngeal compartment plotted as a log-probability function (Minute volume: 20 l/min). (The figure shows that there is a roughly linear relationship between deposition efficiency and the logarithm of the particle size.)

manipulation suggested by the sigmoid shape of the nasopharyngeal and pulmonary deposition curves. The results are shown in Figure 9-4 and Figure 9-5. The deposition in the tracheobronchial compartment is considered, for practical purposes, as constant at approximately 8 percent of the inspired particulate matter.

The results of the Task Group's calculations suggest the type of atmospheric sampling data that will be most meaningful in the correlation of atmospheric particle concentrations and human health. In particular, the insensitivity of the model to aerodynamic size distribution led the Task Group to propose the concept of "respirable"

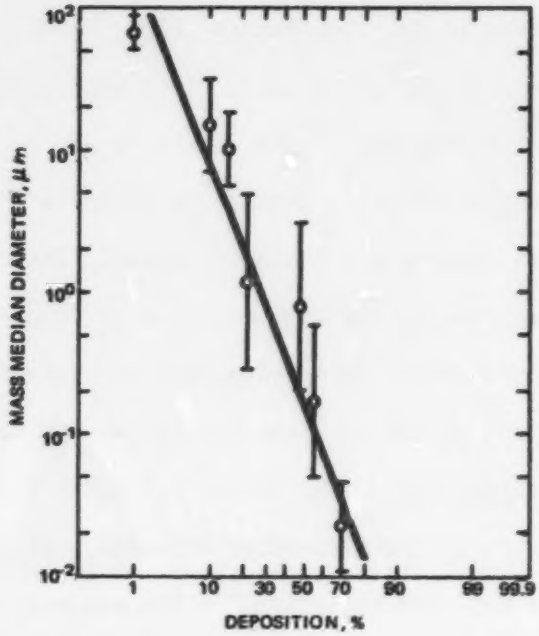

Figure 9-5. Data from Figure 9-3 for the pulmonary compartment plotted as a log-probability function (Minute volume: 20 l/min). (This figure shows that there is a roughly inverse linear relationship between deposition efficiency and the logarithm of particle size.)

dust samples, in which the samplers are designed and calibrated to provide data from which one can determine the mass median diameter in aerodynamic terms. Such considerations have a special significance in connection with particles, such as asbestos, which possess an abnormal deposition behavior.

2. Experimental Studies of Factors Affecting Deposition and Retention

Experimental studies of the deposition of inhaled particulate material may be divided into two broad categories. The first group deals with the measurement of total deposition in the respiratory tract, and the second group is concerned with regional deposition within the various areas of the respiratory tract.

a. Total Deposition in the Respiratory Tract

Experimental studies of deposition were first made by Lehmann et al.,[16] Saito,[17] Owens,[18] Baumberger,[19] and Sayers et al.[20] Then Drinker et al.[21] measured respiratory deposition in man with simultaneous recording of respiratory frequency and minute volume. The concentration of the particles was measured in the chamber, from which air was inhaled, and in the exhaled air. An average retention value of 55 percent at a frequency of 6 to 18 respirations per minute was found.

(1) Effect of Particle Size. The first systematic study of the influence of particle size on the percentage deposition of inhaled dust was made in 1940 by Van Wijk and Patterson.[22] The subjects at rest, inspired mineral dust particles from the air of South African gold mines. Percentage deposition approached 100 percent above 5 μm and had decreased to about 25 percent at 0.25 μm, the limit of the measuring technique.

Brown[23,24] made a series of tests on human subjects in which he found that the percentage deposition is directly proportional to the particle size and to the density of the suspended material. (Size here refers to the physical dimensions of aggregates of particles rather than to unitary particles.)

More sophisticated experiments by Altshuler et al.[25] have used a homogeneous aerosol of triphenyl phosphate with particle sizes in the range 0.14 to 3.2 μm to study deposition in human subjects. It was found that deposition was dependent on particle size and that the minimum deposition diameter was 0.4 μm, which is in reasonable accord with the prediction made by Findeisen (0.6 to 0.8 μm) 20 years earlier.

The deposition of coal dust in human subjects has been shown[26] to rise from a minimum efficiency of about 30 percent at 0.5 μm to almost 60 percent at 0.1 μm (mass median diameter). Such an overall efficiency suggests that the absolute efficiency of deposition of the particles smaller than 0.1 μm in the pulmonary air spaces is close to 100 percent. Direct measurement of the particle concentration in samples of alveolar air showed an efficiency of removal of essentially 100 percent down to about 0.5 μm and better than 80 percent for particles well below 0.1 μm.[27]

A hygroscopic particle may collect sufficient water to increase its size significantly over that in the dry state. Dautrebande and Walkenhorst[26] have therefore compared the deposition of sodium chloride with that of coal dust. Using the dry size of the salt particles, deposition curves different from those for coal dust were obtained, but on correcting (by a factor of 7) to account for the growth of salt to liquid droplets in the respiratory tract, the curves were quite similar for the two particles. This result is of considerable practical significance, as it means that hygroscopic particulate air pollutants will be deposited to a higher degree than mineral particles of an equal size. The Task Group report[4] gives equations which may be applied to correct for the effect of hygroscopicity on deposition.

(2) Physiological parameters. In Brown's experiments,[23,24] the effect of varying respiratory frequency, tidal volume, and minute volume was determined by making measurements on subjects breathing at rest, under various work loads on a bicycle ergometer, and breathing air containing added CO_2. The conclusions of this work were:

a. Percentage deposition is inversely proportional to respiratory rate for rates below 20 per minute and an increase in frequency above 30 per minute causes no further change in percentage deposition;

b. Percentage deposition is inversely proportional to the minute volume; and

c. Percentage deposition is unaffected by tidal volume, vital capacity, or relative humidity of the inspired air.

The results are essentially in agreement with the predictions of Findeisen[12] and Landah[13] (although no effect of tidal volume on deposition was observed).

In general agreement with Brown's result for respiratory rate, Atshuler et al.[25] found in their experiments that slower, deeper breathing gave greater deposition than faster, shallow breathing. It is also shown that, in agreement with prediction, the differences due to respiratory frequency are greater for 1.6-μm particles than for 0.14-μm particles. For the 1.6-μm particles, impaction and settling are the dominant mechanisms of deposition, and the number settling varies as the first power of time. With the 0.14-μm particles, Brownian motion is the dominant deposition mechanism, and the number settling varies as the square root of time.

Experiments with stearic acid particles[28] have revealed an interesting phenomenon of minimal deposition at normal breathing frequencies of 15 to 20 breaths per minute and an increase when the frequencies were either higher or lower than this. A range of respiratory rates from less than 5 to more than 35 per minute was used, and particle diameters lay between 1 and 5 μm. On the other hand, Morrow and Gibb[29] find that the deposition of 0.04-μm sodium chloride particles in dogs and human subjects decreases with an increase in breathing frequency. The deposition percentages themselves (66.5 percent in dogs, 63.4 in man) are close to those predicted by Findeisen. An increase in tidal volume increased the deposition in these experiments in distinction to the absence of a tidal volume effect found by Brown.

There appears to be a direct relationship between percentage deposition and holding time in the lungs for particles of 0.55-μm diameter.[30]

b. Regional Deposition

The experimental study of regional deposition of particles is more complex than is the determination of overall total deposition in the respiratory tract. Various specialized techniques such as the inhalation of radioactive particles followed by external counting over specified portions of the chest, or radioautograph of the lungs, have been employed. The technique of fractionating exhaled air and counting the particles in each fraction has also been used.

(1) Nasal Fractionation. Lehmann,[31] using a test dust of unspecified size, found a median nasal deposition of 46 percent in 185 normal subjects and 27 percent in 241 silicotics. Dust-laden air was blown through the nose and out through a tube in the mouth. Tourangeau and Drinker[32] found deposition efficiencies of 10 to 25 percent with airflow rates through the nose of 4 to 12 l/min. Their dust was calcium carbonate of a size comparable to the silica particles found in silicotic lungs. Reversal of the direction of flow did not alter the values.

The most extensive experimental studies of nasal penetration have been made by Landahl's group.[33,34] The results, especially for corn oil particles, confirm theoretical predictions,[15] and nasal deposition is found experimentally to have a strong dependence on airflow rate.

(2) Lung Deposition. Wilson and LeMer[35] used an aerosol of glycerol containing ^{24}NaCl as a tracer in seven normal subjects who breathed through the mouth at varying frequencies. Particle sizes were in the range of 0.2 to 2.5 μm. External chest counts enabled estimates of lung burden to be made and the pulmonary deposition curve showed a maximum of about 80 percent for particles of about 1.6-μm diameter. A second peak at 0.4 μm was interpreted in terms of the differing optimum sizes for deposition in the finest airways and in the pulmonary air spaces; deposition in these two areas was not distinguished by the method employed.

Indirect estimates of upper respiratory, alveolar, and total deposition in human subjects were made by Brown et al.[36] using a technique for fractionating the exhaled air. Ineach successive portion, the CO_2 content was measured together with the particle count and thus the amount of lung air in each portion of the sample could be estimated. Equal removal efficiencies in both directions were assumed, and a series of expressions was developed for calculating total, alveolar, and upper respiratory deposition. The median particle size of the china clay test dust ranged from 0.24 to above 5 μm, and the total retention decreased systematically from 90 percent or more for particles 5 μm and greater, down to 25 to 30 percent for 0.25-μm particles. Tracheobronchial retention also decreased systematically with particle size but reached zero at a finite size above 1 μm. Alveolar retention, calculated as the percentage of the number of particles reaching the alveoli, remained between 90 and 100 percent for all sizes down to about 1 μm; below that, it decreased in proportion to total retention. The calculated curve for alveolar deposition showed an optimum size at 1 μm.

(3) Comparative Human and Animal Retention. A technique essentially similar to that used by Brown in his experiments on human subjects (see section C-2-a-(2) above) has been employed by Palm, McNerney, and Hatch[37] for studies on guinea pigs and monkeys. This provides a valuable chance to compare results of experiments on laboratory animals and man. The test dusts included china clay, carbon, antimony trioxide, and Bacillus subtilis var. niger. The overall pattern of the results was similar in the experimental animals and in man. The actual deposition and retention values were very close for the monkey and for man. For the guinea pig, total retention was close to 100 percent for 3-μm particles and fell systematically with decreasing particle size. In comparison with man, the total retention was higher, especially in the lower size range. Alveolar retention was essentially

the same in both species, and it is the tracheobronchial retention which is higher in the guinea pig than in man. Although alveolar deposition in the guinea pig was much lower for 1.5-μm particles than in man, because of the removal of these particles in the tracheobronchial tract, the optimum size for alveolar deposition was similar in both species.

3. Conclusions Reached about Alveolar Deposition

Three important conclusions may be reached about the effect of particle size on alveolar deposition:

a. There is a maximum efficiency of deposition at a size between about 1 and 2 μm;

b. There is minimum efficiency for a size of around 0.5 μm; and

c. The percentage of particle deposition for sizes less than 0.1 μm is just as great as for sizes more than 1 μm (Figure 9-3).

This last conclusion is not always given enough weight, even though its prediction by Findeisen[12] has been adequately confirmed experimentally.[26,29] On the other hand, the importance of the second conclusion above should not be overemphasized, as it refers only to the probability of deposition. If their number, and therefore the mass deposited, is relatively great (as may well be for aged aerosols), then particles in the 0.1- to 0.5-μm size region may be as important as smaller and larger particles in provoking toxic response. This is also important when considering particles containing absorbed material.

D. FACTORS AFFECTING THE CLEARANCE OF PARTICULATE MATTER FROM THE RESPIRATORY SYSTEM

A well-known response of a living organism to foreign matter is its attempt to rid itself of, or in some way inactivate, the unwanted material. The overall effectiveness of clearance mechanisms in the lung is well illustrated by the finding that the actual amount of mineral dust found in the lungs of miners or city dwellers at autopsy is only a minor fraction of the total dust that must have been deposited there during their lives. The clearance of certain particles may be very slow. For an individual subject, the rate is dependent upon particle size, site of deposition, and chemical composition.

Relative to other factors, the importance of removal from the respiratory system of trapped particulate materials depends on the rate at which the material elicits a pathological or physiological response. The effect of an irritant substance which produces a rapid response may depend more on the amount of initial trapping than on the rate of clearance. On the other hand, materials such as carcinogens, which may produce a harmful effect only after long periods of exposure, may exhibit activity only if the relative rates of clearance and deposition are such that a sufficient concentration of material remains in the body long enough to cause pathological change. In such a case, the amount of initial deposition will be of relatively minor importance.

Different clearance mechanisms operate in the different portions of the respiratory tract, so that the rate of clearance of a particle will depend not only on its physical properties, such as shape and size, and on chemical composition, but also on the site of initial deposition. Furthermore, the presence of a nonparticulate irritant or the coexistence of a disease state in the lungs may interfere with the efficiency of clearance mechanisms and thus prolong the residence time of particulate material in a given area of the respiratory tract. Kotin and Falk[38] have emphasized the possible importance of this interaction in the pathogenesis of lung cancer (Chapter 10).

1. Clearance Model

The Task Group on Lung Dynamics[4] considered the respiratory clearance of particulate matter, as well as its deposition. The model they developed provides a convenient starting point for a discussion of experimental work on clearance.

Figure 9-6 presents a schematic diagram of all deposition sites and clearance processes.[4] The three conventional compartments of the respiratory tract discussed in connection with the deposition model are used here. D_1 is the particulate material inhaled; D_2 is the material in the exhaled air; D_3, D_4, and D_5 are the particles deposited in the nasopharyngeal, tracheobronchial, and pulmonary compartments respectively, expressed as percentages of D_1 and determinable from the deposition model. In addition to the respiratory tract, three other compartments are listed: the gastrointestinal tract, systemic blood, and the lymph. The different absorption and translocation processes which are associated with the clearance of various compartments are as follows:

a. Rapid uptake of material deposited in the nasopharynx directly into the bloodstream.

b. Rapid clearance of all particulate matter from the nasopharynx by ciliary transport of mucus. This route, and d, have clearance half-times of minutes.

c. Rapid absorption of particles deposited in the tracheobronchial compartment into the systemic circulation.

d. The rapid ciliary clearance of the tracheobronchial compartment. Particles cleared by this route go quantitatively to the gastrointestinal tract.

e. The direct translocation of material from the pulmonary region to the blood.

f. The realtively rapid clearance phase of the pulmonary region dependent on recruitable macrophages. This in turn is coupled to the ciliary mucus transport process for which a half-time of 24 hours has been suggested.

g. A second pulmonary clearance process, much slower than f, but still dependent upon endocytosis and ciliary mucus transport. This process is rate-limited in the pulmonary region by the nature of the particles per se.

h. The slow removal of particles from the pulmonary compartment via the lymphatic system.

i. A secondary pathway in which particles cleared by pathway h are introduced into the systemic blood.

j. The collective absorption of cleared material from the gastrointestinal tract into the blood. No attempt is made to include this factor in the development of the model.

The use of the model requires a knowledge of values for two parameters for each of the pathways just described. These are the amounts of material residing in the compartment which follows a particular exit path (regional fraction), and the rate at which that fraction of the material is cleared (biological half-time) In some cases the kinetic values are physiologically controlled and are more or less independent of the nature of the particulate material. In other cases the physicochemical nature of the deposited material is the critical factor.

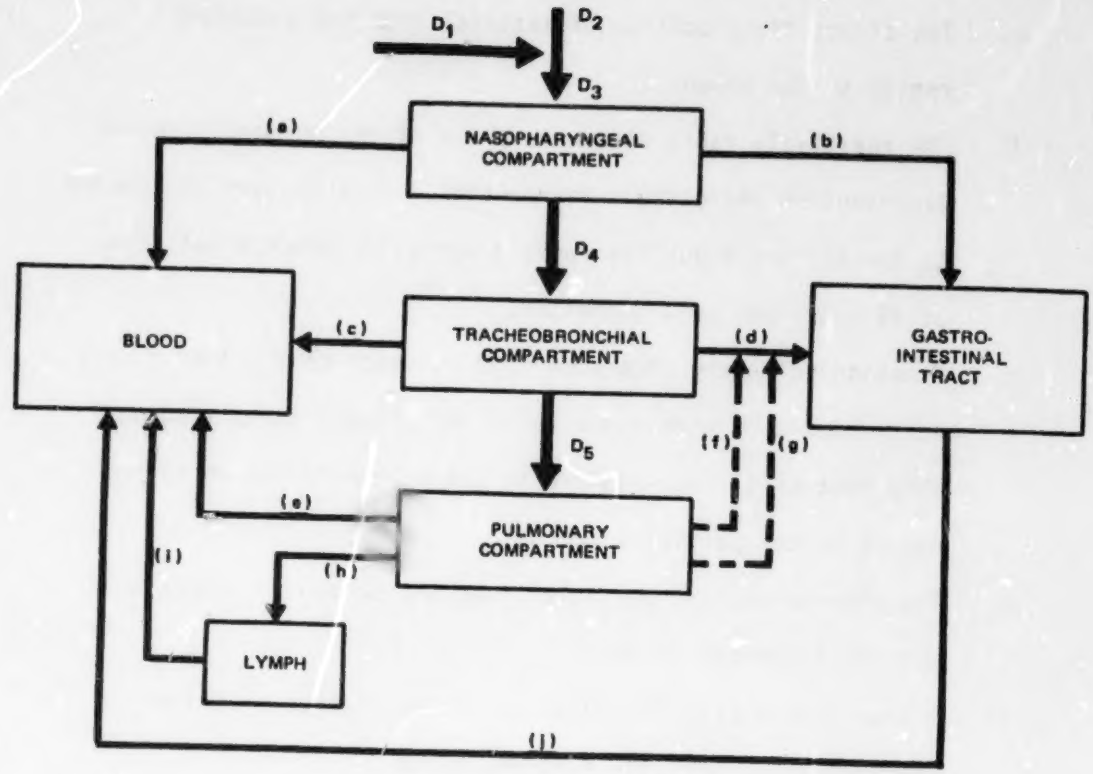
D1
D2
D3
(a)
NASOPHARYNGEAL COMPARTMENT
(b)
D4
BLOOD
(c)
TRACHEOBRONCHIAL COMPARTMENT
(d)
GASTRO-INTESTINAL TRACT
D5
(f)
(g)
(e)
PULMONARY COMPARTMENT
(i)
(h)
LYMPH
(j)

Figure 9-6. Schematic portrayal of dust deposition sites and clearance processes.[4] (This diagram illustrates the various deposition sites and clearance mechanisms used in the model of the lung developed by the Task Group on Lung Dynamics, and described in the text.)

A classification of inorganic compounds is given in the Task Group report, which contains the best available information on both deposition and clearance of inhaled particles.

The Task Group stressed the need for research in the area of solubilities of various compounds in water, in very dilute alkali, and in the presence of proteins. Some of the work done by Morrow et al.[39] correlates clearance of material from the lungs with other properties such as ultrafilterability and clearance from intramuscular injection sites. These data will be of value in understanding the effects of physicochemical properties on clearance.

It will be seen that the clearance mechanisms can be divided broadly into those depending on ciliary action and those which operate in the virtually nonciliated pulmonary region. The two sections which follow describe experimental work concerned with these two general subdivisions.

2. Clearance from the Tracheobronchial System

A blanket of mucus in the tracheobronchial region is kept in continual upward movement by the ciliary activity of the columnar epithelium lining which extends down as far as the terminal bronchioles. The frequency of the ciliary beat has been found[40,41] to be 1,300/min in the rat, while the overlying mucus fluid was found to move at an average rate of 13.5 mm/min. Similar transport rates of 15 mm/min in excised trachea and 18 mm/min in intact animals have been reported by Antweiler.[42] In this latter study, several particulate materials were used, including soot, coal dust, lycopodium spores, cork dust, aluminum powder, and glass and lead spheres. Transport rates seemed to be unaffected by size, weight, or shape of the particles except in the case of the glass spheres, which were said to be sufficiently smooth to allow the mucus to flow over them rather than to transport them.

It has been demonstrated[43,44] that in the human lung the mucus flows over only 10 to 20 percent of the theoretically available surface at the dividing passages where the airways branch. The mucus blanket divides and flows in two directions around the margins of the opening. This phenomenon, taken together with the greater probability of impact deposition at bifurcations, may explain why histological sections show accumulations of particles at bronchial branch points.[45]

In studies of respiratory system clearance, use has been made of monodisperse aerosols tagged with radioactive substances to permit the subsequent fate of the material to be followed by external counting techniques. In general, the results indicate that clearance occurs in two distinct phases of about 2- and 10-hour durations, probably representing the clearance of particles from the proximal and distal parts of the bronchial tree. Albert et al.[46] studied clearance from the human lung of 3- and 5-μm iron oxide particles tagged with ^{51}Cr and ^{198}Au. The rapid phase of clearance was completed within one day, and in most cases within 12 hours. The clearance curves obtained in these studies did not differ substantially from those obtained earlier by Albert and Arnett[47], who used a heterogeneous iron oxide aerosol, suggesting that the actual distribution function of particle sizes may be of little importance in determining clearance rates. However Holma[48] generated a bidisperse aerosol of 6- and 3-μm polystyrene particles, tagged respectively with ^{198}Au and ^{46}Sc, and measured simultaneous clearance of the two sizes in rabbits. The larger particles cleared more rapidly than the smaller ones. The mean half-life for the initial phase was 0.48 hours for the 6-μm particles and 1.07 hours for the 3-μm particles. The respective half-lives for the long clearance phase were 69.7 and 210 hours.

The effect of irritant gases on clearance of particles is obviously of significance where air pollution involves this combination of factors. Dalhamn's[40, 41] studies showed that the acute response to irritant gases such as ammonia, formaldehyde, or surfer dioxide was a cessation of ciliary beat. The time to cessation was dose related. Chronic exposure of rats to one of the irritants sulfur dioxide slowed down or caused a complete cessation of the transport of tracheal mucus, but the average beat frequency of the cilia was the same as in normal animals. The cessation of clearance was a result of an increase in the thickness of the mucus layer of from 5 to 25 μm.

The material carried upward by ciliary action is swallowed and thus enters the gastrointestinal tract. Brieger and LaBelle[49] exposed animals to a water-insoluble dye and demonstrated that 24 hours after the termination of exposure, over 50 percent of the total dye found in the body was in the intestinal tract. This phase persisted for several days, during which time a significant intestinal burden was present. The concentration of dye finally fell to very low values after a week or so, and most of the dye that remained in the body was found in the lung, indicating that the rapid phase of ciliary clearance was finished. It has also been shown[50] that uranium dioxide, cleared from the respiratory system to the gastrointestinal tract by ciliary action, is responsible for the high urinary uranium levels seen during the first days after exposure to uranium dioxide dust. The possibility of consequences of relatively high concentrations of an atmospheric pollutant appearing in organs remote from the lungs must not be overlooked. In this connection, the high incidence of stomach cancer in areas suffering from high levels of atmospheric pollution takes on a particularly ominous appearance.[51]

3. Clearance from the Alveolar Surface

Particles deposited on the alveolar surface may be removed by any of the mechanisms (e) to (i) given in the clearance model of section D-1, and they may also become sequestered by a tissue reation within the lung (pneumoconiosis), or become bound to protein material in the lungs. Of the several clearance mechanisms, the most rapid is that involving recruitable macrophages (phagocytic cells contained on the alveolar surface epithelium). Phagocytosis may also serve to render the particles incapable of injuring or irritating the alveolar surface epithelium and may to some extent prevent the penetration of the particles into the interstitium of the lung. The origin and behavior of these alveolar macrophages have been the subjects of active research in recent years.[45, 51-53]

LaBelle[54, 55] and LaBelle and Brieger[55] demonstrated that the clearance of particulate matter by phagocytosis can be markedly influenced by the dust load presented to the lung. The initial observation was made that clearance curves obtained with microgram quantities of activated uranium dioxide were noticeably different from those obtained earlier using milligram amounts. When carbon particles were added to the activated uranium dioxide to bring the total weight of administered dust into the range of the earlier studies, the clearance curves were quite similar. In seeking the reason for this finding, LaBelle demonstrated that the number of free phagocytes washed out of the lungs was related to the dust load and that the amount of dust eliminated from the lungs during the early postexposure period was proportional to the number of free phagocytic cells present. Within the limits of experimental error, the kinetics of elimination of particles and the kinetics of the disappearance of the phagocytic cells following exposure were identical for both inhalation and intratracheal exposures.

The relationship between concentrations in the different respiratory regions during alveolar clearance has been the subject of a short-term study by Gross, Pfitzer, and Hatch.[57] The clearance of four kinds of dust burden (antimony trioxide, ferric oxide, quartz, and coesite) was followed in rats whose lungs had been loaded using the inhalation and intratracheal injection techniques. Initially, although the greater concentration of dust was to be found in the proximal alveoli, dust deposition was also prominent in alveoli distal to alveolar ducts. However, within 3 or 4 days, the dust in the distally situated alveoli had largely disappeared and had apparently become concentrated in the proximal alveoli. This stagnation of dust in the evaginating alveoli of the respiratory bronchioles and alveolar ducts may help to explain the greater vulnerability of these regions to inhaled irritants.

E. SUMMARY

The respiratory system may be divided into three sections--nasopharyngeal, tracheobronchial, and pulmonary systems. Deposition and clearance mechanisms may differ for the various parts of the respiratory tract. A particle of any size which passes the nasopharyngeal region may be deposited in the remainder of the respiratory tract and, although the actual mechanism of deposition is primarily dependent upon the particle size, the shape of the particle can also affect the efficiency of its deposition. Consequently, if atmospheric dust loads are to be related quantitatively to health hazards, the dust samplers used for monitoring should have collection characteristics similar to or the same as the human lung. The fast phases of the lung clearance mechanisms are different in ciliated and nonciliated regions. In ciliated regions, a flow of mucus transports the particles to the entrance of the gastrointestinal tract, while in the nonciliated pulmonary region phagocytosis by macrophages can transfer particles to the ciliated region. The rate of clearance is an important factor in determining toxic responses, especially for slow-acting toxicants such as carcinogens. In addition, since the clearance of particles from the respiratory system primarily leads to their entrance into the gastrointestinal system, organs remote from the deposition site may be affected. The models developed by the Task Group on Lung Dynamics are used in this chapter as a basis for discussion of experimental data on the deposition, retention, and clearance of particles. These models provide a useful representation of the deposition and clearance mechanisms and have been shown to yield predictions which have often been substantiated by experimental findings.

F. REFERENCES

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Chapter 10

TOXICOLOGICAL STUDIES OF ATMOSPHERIC PARTICULATE MATTER

A. INTRODUCTION

Experimental toxicology, using specific atmospheric pollutants, would be the best means for deriving air quality criteria, provided that man could be used as the experimental animal. However, the ethical imposibility of performing experiments using human exposures to varying concentrations of a wide range of compounds precludes so direct an approach. Although a limited amount of intentional human experimention may be possible, most of the data for human toxicology are derived from accidental or occupational exposures. The use of laboratory animals in toxicological experiments is more straightforward, but the obvious anatomical and metabolic differences between the animals and man require the exercise of considerable caution in applying the results of animal exposures to human health criteria. Furthermore, many of the animal experiments performed have left something to be desired in terms of air pollution toxicology, since the "endpoints" of the experiments have frequently been the deaths of the animals at exposures to concentrations far in excess of those likely to be found, or tolerated, in the atmosphere. There is a great need for chronic inhalation studies to define long-term effects occurring over a lifespan.

These difficulties and limitations of toxicological studies should not be obscure the fact that an increasing amount of data useful for air quality criteria is being amassed, it is, however, always essential to bear in mind the limitations of the data. Toxicological studies have shown that atmospheric particles may elicit a pathological or physiological response in at least three ways. First, the particle may be intrinsically toxic; second, the presence of an "inert" particle in the respiratory tract may interfere with the clearance of other airborne toxic materials, and third, the particle may act as a carrier of toxic material. There is also evidence that the presence of particles may occasionally reduce the toxicity of a second pollutant; this phenomenon is described briefly. From an air pollution standpoint, one of the most ubiquitous particulate pollutants is smoke; some pathological studies involving this material are presented. Studies of physiological response to irritant particles and to mixtures of particulate matter with irritant gases are described.

A final section of this chapter then discusses carcinogenesis and atmospheric pollutants.

B. MECHANISMS OF TOXICOLOGICAL ACTION OF PARTICULATE MATTER

1. Intrinsic Toxicity

A few common atmospheric particulate pollutants appear to be intrinsically toxic, of these, the most important is sulfur trioxide (either as the free oxide or hydrated as sulfuric acid, H_2SO_4), which has a high degree of toxicity, at least for the guinea pig. Although silica (from fly ash) is frequently present as a pollutant, atmospheric concentrations are normally too low to lead to silicosis. In recent years, however, concern has been expressed over a number of less common toxic particulate pollutants, including lead, beryllium, and asbestos. Increasing amounts of lead (as oxides and salts) are being discharged into the atmosphere as a result of the burning of gasoline containing lead additives, on the other hand, other sources contributing to atmospheric lead seem to be decreasing and, according to Stokinger and Coffin,[1] toxic effects due to lead do not seem to present a serious risk to the population at large. Recent reviews have shown that the population at large has remained in lead balance even in the face of continued and increasing burning of leaded gasoline. Beryllium (as the oxide BeO) can lead to chronic pulmonary disease with a high fatality rate. Accidental exposures have shown that there may be a considerable latency period before disease develops, and some alarm has been generated over the use of beryllium as a rocket fuel component, although the rocket effluent is predominately a "high-fired," relatively inert form of beryllium oxide. The situation with regard to asbestos is also potentially serious. Brief industrial or accidental exposures to asbestos can lead, after a latency period of 40 years or more, to the development of diffuse mesotheliomas of the pleura or peritoneum, and it is possible that sufficiently high concentration of asbestos might be generally present in the atmosphere to constitute a definite health hazard. This possibility is made more likely by the greatly increased use of asbestos after the occurrence of the exposures which are only now leading to the development of mesotheliomas. Human mesothelioma is generally associated with inhalation of crocidolite, but chrysotile and amosite can induce this tumor also.[2] Thus, there can be little doubt that exposure to asbestos is the major factor in the causation of this tumor.[3] A series of autopsies has shown that an appreciable fraction (20 to 50 percent) of the population at large has "asbestos bodies" in its lungs. More detailed discussions of both asbestos[4] and beryllium[5] toxicity may be found in recent reviews.

Several other potential carcinogens are known to be present as relatively minor atmospheric pollutants; these may be particulate in nature, and they are discussed in section D-1.

2. Adsorbed Substances

Toxic substances may be adsorbed on the surface of particulate matter, which may then carry the toxic principle into the respiratory system. The presence of carbon or soot as a common particulate pollutant is noteworthy, as carbon is well known as an efficient adsorber of a wide range of organic and inorganic compounds. Some specific studies involving mixtures of particles and irritant gases are presented in section C-3, the present section is concerned mainly with the general principles of adsorption and desorption.

The role played by the affinity for the adsorbate by the particle is complex. A high affinity will mean that relatively large laods of adsorbate may be carried by each particle. If the adosrbate in its free state is slowly removed from the air in the respiratory system, then the deposition of particles carrying high concentrations may constitute a greater toxic hazard, especially at the localized deposition points. Whether or not the effect is significant depends on the efficiency of the desorption and elution process relative to that of the clearance process. The chemical nature of both adsorber and adsorbate, and the size of the adsorbing particle, all play a part in determining these various efficiencies, and each system will show its own individual characteristics. Carcinogens, which may produce their effect only after long or repeated exposure, present a particularly involved situation, because it is not clear whether slow release of small concentrations of the carcinogen is more dangerous than the rapid release of larger quantities. Experimental evidence on the elution of a specific series of carcinogens is described in section D-2.

3. Reduction of the Toxicity of Irritant Gases

The finding that a preexposure to airborne particles will tend to protect animals against the action of an irritant gas is not an uncommon one.

Pattle and Burgess[6] found that, with mice and guinea pigs, the previous inhalation of smoke reduced the toxicity of sulfur dioxide (measured in terms of the dosage required to produce death). They postulated that the reduced toxicity of SO_2 produced by preexposure to smoke was due either to the action of the smoke in reducing the volume breathed (and thus the SO_2 dose received), or to a stimulation of secretions which may protect the mucosa from the irritant action of the gas. This explanation is given weight by their findings that increased toxicity resulted from the administration of mixtures of smoke and sulfur dioxide. A similar study by Salem and Cullumbine[7] indicated that the effect of kerosene smoke on the toxicity of irritant substances depended to some extent on the species of animal, although the toxicity of acrolein and acetaldehyde seemed to be decreased in most cases.

Wagner et al.[8] observed that the preexposure of mice to oil mists would protect against the later action of inhaled ozone and nitrogen dioxide. The greatest protection was obtained when the oil mist was inhaled 18 hours prior to the exposure to the irritant gas; when the oil mist and irritant gas were administered together, there was an enhancement of toxicity. The relative degree of this enhancement was consistent with the known depth of penetration of nitrogen dioxide and ozone in the respiratory tract and the relative solubility of the two gases in mineral oil. The hypothesis for the protective action of the pretreatment with oil is the formation of an oil film on the alveolar surface.

Again, Amdur and Devir,[9] using guinea pigs, have observed that the presence of an aerosol of 2.5 μm triphenyl phosphate particles at a concentration of 50 to 100 mg/m^3 lessens the increase in pulmonary flow resistance due to inhalation of sulfur dioxide. They also studied the effect of treatment with the particulate material before exposures to sulfur dioxide alone. This sequence afforded excellent protection against the response to the sulfur dioxide, and the degree of protection was related to the total amount of aerosol inhaled.

C. TOXICOLOGICAL STUDIES OF SPECIFIC PARTICULATE MATERIALS

1. Pathological Studies of Smoke and Carbon Particles

Smoke from burning bituminous coal was used for a study on rabbits and rats by Schnurer and Haythorn.[10] Four rabbits and eight rats were exposed for periods of 80 days to smoke with a concentration of 4,410 particles/cm^3 (125 million/ft^3), of which 8 percent was free SiO_2. Control animals received clean filtered air. One control rabbit and two exposed rabbits died of bronchial pneumonia before the end of the exposure. One rabbit and six rats were autopsied immediately after exposure, and the rest at intervals up to 429 days. Lesions typical of nonoccupational anthracosis were noted in the lungs of the animals killed immediately after exposure, while fibrous reactions developed around the carbon deposits (with the formation of collagen strands) in the animals that were examined several months to a year after the exposure. The authors state that these lung changes were analogous to those seen in a milder grade of bituminous pneumoconiosis of soft-coal miners, and they also concluded that the pneumonitis and fibrosis were attributable to the carbon rather than to the small amount of silica present.

Schnurer[11] has also attempted to compare the response to the burning of equal weights of anthracite coal, coke, and bituminous coal. Unfortunately, widely differing particle concentrations were obtained from burning equal weights of fuel, so that it is impossible to place any simple interpretation on the results of the experiments.

Pattle et al.[12] studied the acute toxic effects of smoke, generated by burning tetrahydronaphthalene, in the concentration range of 700 to 1,100 mg/m^3. They found that the median dosage to death for guinea pigs and mice lay between 147,000 to 351,000 mg-min/m^3. In mice, the causes of death were blockage of the air passages, and delayed deaths resulting from the exposure were unusual. The guinea pigs showed hemorrhagic lesions, and delayed deaths were more common. The action of smoke on rats resembled that on mice. No data are provided to show that unburned tetrahydronaphthalene did not enter the smokestream in these experiments; toxic effects are well known for this substance.

In the course of a study of mixtures of sulfur dioxide and smoke, to be discussed in section C-3, Pattle and Burgess[6] reported some data on the effect of smoke alone on mice. The smoke was generated by a burning kerosene lamp, and its concentration was about 50 mg/m^3. Exposure was continuous for 36 hours, followed by an interval of 11 hours of unpolluted air. Smoke exposure was then administered for an additional 30 hours. There were no fatalities and none occurred after the experiment. Histological examination of the lungs showed that at the end of exposure the soot particles were partly spread over the lining of the bronchioles and alveoli and partly aggregated into patches.

There were no signs of edema, consolidation, hemorrhage, or emphysema, and capillary congestion was slight. The lungs were normal except for the presence of carbon particles, and animals killed 3 months after exposure showed phagocytosis and gradual clearance of smoke from the lungs.

Salem and Cullumbine[7] also make brief mention of the exposure of mice and guinea pigs to smoke from a kerosene lamp. The animals survived exposure to 664 mg/m^3 of smoke for 6 hours, and autopsy revealed no obvious damage to the lungs.

The physiological effects on mice of exposure to carbon black by ingestion, skin contact, subcutaneous injection, and inhalation were examined by Nau and his coworkers[13-16]. Channel black (particle diameter 0.025 μm) and furnace black (particle diameter 0.035 μm) were used in concentrations of 2.4 and 1.6 mg/m^3 respectively for the inhalation experiments. Mice, hamsters, guinea pigs, rabbits, and monkeys were exposed for prolonged periods to the dust, but no effect other than the accumulation of carbon particles in the lungs was demonstrated.

The problem of whether exposure of rats to coal dust or smoke would alter their susceptibility to infection by Type I pneumococci was examined by Vintinner and Baetjer[17] as part of a series of studies on the effects of fibrous, inert, and adsorptive dusts on susceptibility to infection in experimental animals.[18-20] The concentration of coal dust varied between 14 and 30 thousand particles/cm^3 (between 400 and 850 million particles/ft^3) with an average of 25 thousand particles/cm^3 (700 million particles/ft^3). The smoke level in the chambers would have been considered as "dense" on visual inspection, or equivalent to about a

Number 3 reading on the Ringelmann Chart. Analyses of the particulate matter in milligrams per cubic meter in the smoke chamber averaged as follows: total solids-570, carbon-470, ash-93, silica-46, iron-15, and sulfur-9. The exposures ranged from 5 to 165 days for the bituminous coal dust and from 2 to 154 days for the smoke. On the basis of their extensive data, the authors concluded that the inhalation of smoke did not alter the susceptibility to infection when the organisms, either in broth or in mucin, were administered by intrabronchial injection. The bituminous coal dust seemed to exert a slight protective effect when the organisms were suspended in mucin but not when they were injected in a broth medium.

Based upon the reported studies, smoke or carbon black on its own apparently produces little major damage to the respiratory system of animals even at exposure levels some orders of magnitude greater than those encountered in polluted atmospheres.

2. Physiological Studies of Responses to Particulate Material

Certain particulate materials are pulmonary irritants which have been shown to produce alteration in the mechanical behavior of the lungs, the alteration being predominantly an increase in flow resistance. This was demonstrated by Amdur[21] for sulfuric acid, and by Amdur and Corn[22] for ammonium sulfate, zinc sulfate, and zinc ammonium sulfate, using the guinea pig as an assay animal. (The decreased flow rates observed by Amdur, Silverman, and Drinker[23] in human subjects exposed to sulfuric acid mist probably reflect an increase in flow resistance, although, since the resistance was not measured in those experiments, such a statement cannot be conclusive.) A series of papers making use of the increase in pulmonary flow resistance as an assay tool has been published by Amdur and Underhill[24] and by Amdur,[25-29] and "response" to an irritant in discussion of these works generally refers to this increase.

Nadel et al.[30] report a correlation between the alterations in pulmonary mechanics and actual anatomical change in cats exposed to aerosols of histamine and zinc ammonium sulfate. The authors discuss the physiological mechanisms which may operate to bring about such alterations in pulmonary mechanics.

In connection with a study of the effect of various aerosols on the increase in pulmonary flow resistance in guinea pigs produced by sulfur dioxide, Amdur and Underhill[24] first examined the response to the aerosols alone. These aerosols included spectrographic carbon at 2 and 8 mg/m^3, activated carbon at 8.7 mg/m^3, manganese dioxide at 9.7 mg/m^3, open-hearth dust at 7.0 mg/m^3, iron oxide Fe_2O_3) at 11.7 and 21.0 mg/m^3, manganous chloride and ferrous sulfate at 1 mg/m^3, and sodium orthovanadate at 0.7 mq/m^3; the particle sizes were less than 0.5 μm. Standard 1-hour exposures were used in this routine bioassay method, with the exception of the iron oxide exposures, which were extended to 2 hours. In no instance did any of the aerosols produce an alteration in flow resistance. The failure of the aerosols to produce an increase in resistance in these experiments suggests that if they cause bronchoconstriction, it must be only in extremely high concentrations. From the point of view of air pollution toxicology, none of them would be classed on this basis as pulmonary irritants. An uncharacterized "fly ash" from an oil-fired burner was also tested and proved inert.

At a concentration of 1 mg/m^3, ferric sulfate aerosol produced a 77-percent increase in flow resistance in a group of 15 animals which was statistically significant at the level of $P<0.001$. Ferric sulfate, in distinction to ferrous sulfate, must be classed as an irritant.

On the other hand, Dautrebande[31] and DuBois and Dautrebande[32] have reported constriction and increased airway resistance in isolated guinea pig lungs and in human subjects with a wide variety of supposedly "inert" particulate matter. The relationship of their results to Amdur's work is not clear since Dautrebande's particle concentrations appear to be abnormally high. Total lung capacity and vital capacity were not altered by the inhalation of particulate material in healthy subjects, but the vital capacity in patients with pulmonary disease was reduced by the inhalation of particulate materials. It is interesting in this connection that guinea pigs with an initially high pulmonary flow resistance showed a greater response to low concentrations of irritant aerosols or of inert aerosol-irritant gas combinations than animals with average control flow resistance values.[33]

The possibility that the conflict between Amdur's and Dautrebande's work may be resolved in terms of the doses involved is given credence by studies which suggest that human response to coal dust may be dose related.[34] Coal dust clouds similar to those in mines have been produced in the laboratory, and the number and weight of respirable particles have been measured. Clouds containing 8, 9, 19, 33, and 50 mg/m^3 of dust in the size range of 1 to 7 μm were used. Normal subjects inhaled the dust for 4 hours, and airway resistance was measured with a body plethysmograph. No changes were obtained after the inhalation of coal dust from clouds containing 8 or 9 mg/m^3, but with concentrations of 19, 33, and 50 mg/m^3, significant increases in airway resistance occurred and the response increased with the dust concentration. One hour after exposure ceased, the airway resistance was about two-thirds back to normal. With the two highest concentrations, the respiratory rate increased throughout the 4 hours, and subjects complained of difficulty in breathing after 1 to 2 hours. It would appear that if 8 or 9 mg/m^3 produced no lung function change in a 4-hour period, the material (coal dust) and perhaps other "inert" particulate matter would be unlikely to do so at any concentrations likely to occur in air pollution.

Particle size may play an important part in determining the potency of an irritant. For example, at a mass concentration of 1.4 to 1.9 mg/m^3, sulfuric acid mist of 0.8 μm produces a 51-percent increase in pulmonary flow resistance in guinea pigs compared to control values,[21] and zinc ammonium sulfate particles of 0.84 μm produce a 21-percent increase.[27] If the zinc ammonium sulfate size is 0.3 μm, the corresponding increase in pulmonary flow resistance is 130 percent.[22] The studies on zinc ammonium sulfate were carried out by Amdur and Corn[22] with nonhomogeneous aerosols with mean sizes by weight of 0.24, 0.51, 0.74, and 1.4 μm, at several concentrations. As the particle size decreased over the range 1.4 to 0.24 μm, the response to an equal mass concentration rose. When dose-response curves of percent increase in flow resistance against concentration in milligrams per cubic meter were plotted for the different particle sizes, the slopes increased as the particle size decreased. A similar study was undertaken by Amdur and Creasia[35] using m-terphenyl as the aerosol (size range 0.3 to 2.0 μm); this aerosal did not absorb water during passage through the respiratory tract. An essentially identical pattern to that seen for zinc ammonium sulfate emerged, with the irritant potency increasing with decreasing particle size and the slopes of the dose-response curves becoming steeper for the smaller particles. All of the particles used in these studies fall within the "respirable size range." The possible implications for air pollution are:

a. Particles below 1 μm may have greater irritant potency than larger species, and

b. A small increase in concentration could produce a greater-than-linear increase in irritant response when the particles are smaller than 1 μm.

The effect of particulate matter on clearance mechanisms has already been mentioned. The deposition of particles affects mucous secretion and ciliary action most markedly at branchings of the tracheobronchial tree. Here alterations in the morphology are initially most intense and prolonged and, ultimately, are irreversible. The adverse effect is manifested by a slowing of the flow of the mucous stream, alterations in the physical and chemical properties of mucus, and changes in ciliary action.

Examination of sections of respiratory epithelium removed from the lungs revealed hyperplastic and metaplastic changes earliest at these sites. The exaggerated effect of the impingement of irritants on respiratory epithelium at branch points taken together with less efficient clearance is consistent with this result. Thus, abnormal retention and accumulation of soot (and possibly carcinogenic particles) may occur, especially in segmental bronchi. The accompanying peribronchial and peribronchiolar inflammatory responses further interfere with physiologic mechanisms of defense.

Experimental confirmation of enhanced retention in the presence of irritant material is to be found in the work of Tremer et al.[36], who exposed rabbits first to synthetic smog and then to soot. The results are shown in Table 10-1.

The main conclusions of this section are:

a. The predominant physiological effect of irritants is to increase pulmonary flow resistance,

b. Exceptionally heavy loads of relatively inert particles may cause some increase in flow resistance, and

c. The intensity of physiologic response may increase with a decrease in particle size for any given irritant.

3. Experimental Studies of Mixtures of Irritant Gases and Particles

The possible influence of inert particles on the toxicity of irritant gases has been the subject of considerable speculation[37-39] and a limited amount of experimental work. Knowledge of such interaction of

Table 10-1. EFFECT OF EXPOSURE OF RABBITS TO 2 PPM OZONIZED GASOLINE ON RETENTION OF INHALED SOOT.[36]

Specimen	Smog exposure, hours	Room air (recovery), hours	Soot exposure, hours	Room air (recovery), hours	Soot in lung, mg
No recovery period after smog exposure:					
Control	0	0	1	0	3.7
Animal No. 1	1	0	1	0	7.9
Animal No. 2	1	0	1	24	5.4
With varying recovery periods:					
Control	0	0	1	0	1.1
Animal No. 1	1	4	1	0	7.4
Animal No. 2	1	8	1	0	5.2
Animal No. 3	1	24	1	0	2.0

gaseous and particulate pollutants might be important to understanding the complicated toxicological picture of the air pollution disasters.

The first experimental evidence that an inert aerosol could alter the response to an irritant gas was presented in 1939,[40] when it was reported that concentrations of mustard gas which were relatively harmless to rats would produce pulmonary edema and death when administered in combination with an inert aerosol (sodium chloride). It was postulated that the adsorption of gas on the particles had increased the amount of irritant vapor reaching the critical target areas of the lung. Dautrebande and his coworkers[41,42] studied the sensory responses of human subjects to pollutants thought to be constituents of the Los Angeles smog and found that the presence of particles of sodium chloride, oil mist, or smoke do increase the irritation of eyes, nose, and throat by sulfur dioxide, formaldehyde, and other gaseous pollutants.

LaBelle et al.[43] studied the effect of particulate matter on the survival time of mice exposed to formaldehyde, acrolein, and oxides of nitrogen. The particles used included triethylene glycol, ethylene glycol, mineral oil, glycerin, sodium chloride, two commercial filter grades of diatomaceous earth, and a commercial silica gel. From theoretical considerations, the probable percentage penetration of the upper respiratory tract by various gases was calculated. If the gas penetrated to a greater extent than the particles, then adsorption on the particles would decrease the amount of irritant gas reaching the lungs and the toxicity would decrease. Such a situation exists with oxides of nitrogen. Conversely, if the gas did not readily penetrate the upper respiratory tract, sorption on small particles would tend to carry more gas to the lungs and thus increase the toxicity. Such a situation exists with formaldehyde. The theoretical calculations coincided with experimental results in over 70 percent of the gas-particle combinations. The theory partially explains the potentiation of irritant gases by particulate material (often termed a synergistic effect), but subsequent research has shown that the problem is not as simple as had been assumed.

A synergistic effect was reported by Dalhamn and Reid[44] with ammonia and carbon particles. Ammonia is a highly soluble gas, so that synergism would have been predicted by LaBelle et al.[43] Rats were exposed for 60 days to 68 mg/m^3 (100 ppm) ammonia alone, 7 mg/m^3 carbon alone, and to 86 mg/m^3 (119 ppm) ammonia plus 3.5 mg/m^3 carbon. The carbon particles were 95 percent smaller than 3 μm and 65 percent smaller than 1 μm. In the group exposed to both ammonia and carbon, there was a high frequency of mucosal damage, and the ciliary activity seemed to be significantly impaired. The tracheas of rats exposed to ammonia alone showed less severe damage, and the tracheas were histologically normal in about 80 percent of the rates exposed to carbon alone.

Boren[45] exposed mice to carbon alone, to nitrogen dioxide, and to carbon which had previously been exposed to nitrogen dioxide. He stated that around 550 mg of nitrogen dioxide were adsorbed per gram of carbon. Samples of air taken from the chamber during the exposure of mice to carbon with adsorbed nitrogen dioxide indicated that there was about 47 to 56 mg/m^3 (25 to 30 ppm) free nitrogen dioxide. Although this is a high concentration of nitrogen dioxide, mice exposed to even higher concentrations 470 mg/m^3 (250 ppm) of nitrogen dioxide alone developed pulmonary edema, but neither single nor repetitive exposure produced parenchymal lung lesions. Control mice and mice exposed to carbon alone showed no anatomical abnormality of the lungs. Mice exposed to the carbon with adsorbed nitrogen dioxide developed focal destructive pulmonary lesions. The exposures in this group were 6 hours per day, 5 days a week, for 3 months.

The typical lesions of pneumonitis were observed by Gross et al.[46] in the lungs of hamsters, rats, and guinea pigs exposed to a "sufficiently large number of carbon particles with either adsorbed sulfur dioxide or nitrogen dioxide." Exposure was 8 hours per day, 5 days per week, for 4 weeks. The particle size of the carbon and the meaning of the phrase "a sufficiently large number of carbon particles" were not given. There was no record of the amount of either gas adsorbed by the activated carbon particles. Histological sections were from animals killed about a month after the end of exposure, whereby it is concluded that the lesions were persistent. They were concentrated in the regions of the respiratory bronchioles and alveolar ducts and consisted of cellular wall thickening.

Pattle and Burgess[6] studied the effect of mixtures of sulfur dioxide and smoke on mice and guinea pigs. Their concentrations of sulfur dioxide were in the range of 2,700 to 12,000 mg/m^3 (900 to 4,000 ppm), and the smoke concentrations were in the range of 50 to 135 mg/m^3. The experiment end point was the dosage required to produce death. With concentrations of this magnitude, the results obtained have little applicability to air pollution criteria. Although they found that the lethality of mixtures of sulfur dioxide and smoke was greater than the lethality of the sulfur dioxide alone, they considered the effect to be a simple additive--one resulting from the action of smoke in blocking the bronchi and alveoli.

Salem and Cullumbine[7] studied the effect of kerosene smoke on the acute toxicity of sulfuric acid, sulfur dioxide, acrolein, and acetaldehyde in guinea pigs, mice, and rabbits. As in the work by Pattle and Burgess,[6] the concentrations were many magnitudes above those found in air pollution episodes. The administration of smoke prior to the exposure to the irritant substances did not alter the toxicity, although the effects of smoke on the toxicity of the irritants were highly variable when the two agents were given simultaneously. In guinea pigs, the toxicity of sulfuric acid was increased by the presence of smoke, the toxicities of acetaldehyde and sulfur dioxide were decreased, and the toxicity of acrolein was unchanged. In mice, the toxicity of sulfur dioxide was increased, while those of acetaldehyde and acrolein were decreased. In rabbits, the toxicities of acrolein and acetaldehyde were decreased by the smoke. The end point in all cases was the mean fatal dose.

A series of studies of the effect of hygroscopic particles on physiological response to irritants has been undertaken by Amdur and Underhill[24] and by Amdur,[25-29] using the pulmonary flow resistance technique. It was found initially that the response to sulfur dioxide was increased by particles of sodium chloride below 1 μm but not by 2.5-μm particles, at concentrations of about 10 mg/m^3. With sulfur dioxide[26] and with formaldehyde[27] as irritants, decreasing the concentration of the aerosol, or the total dose of aerosol by shortening the exposure time,[24] decreased the degree of potentiation observed from the addition of sodium chloride. The hypothesis of LaBelle et al.[43] that irritant gases with high water solubility would be potentiated by particles did not explain adequately the data obtained. Sulfur dioxide, formaldehyde, acetic acid, and formic acid all have high water solubility, but it was found that the first two were potentiated by sodium chloride[25,26] and that the latter two were not.[27, 29] Although both sulfur dioxide and formaldehyde were potentiated by sodium chloride, there are differences which suggest that the guiding mechanism in the case of sulfur dioxide may be chemical change and for formaldehyde may involve surface adsorption.[26]

Another paper[24] examines further the effect of various physical and chemical factors on the potentiation of sulfur dioxide by particulate material. The degree of potentiation observed could be correlated to some extent with the solubility of sulfur dioxide in the solutions of sodium chloride, potassium chloride, and ammonium thiocyanate.

Aerosols of soluble salts of ferrous iron, manganese, and vanadium, which had been shown by Johnstone and coworkers[47, 48] to be capable of catalyzing the conversion of sulfur dioxide to sulfuric acid when they were present as nuclei of fog droplets, showed a major potentiating action where present at concentrations of about 1 mg/m^3. On the other hand, dry manganese dioxide, activated or spectrographic carbon, iron oxide fume, open-hearth dust, and triphenyl phosphate did not alter the response even when present in concentrations of 8 to 10 mg/m^3. Similar results have been obtained with iron oxide and redispersed open-hearth dust at levels of 7 to 20 mg/m^3 in guinea pigs. Addition of SO_2 at levels of 216 to 432 mg/m^3 (0.08 to 0.16 ppm) gave results corresponding to gas exposure alone.[49]

It is clear from the data presented[24] that all particulate material does not potentiate sulfur dioxide any more than one compound (sodium chloride) potentiates all irritant gases tested. Both solubility of sulfur dioxide in a droplet and catalytic oxidation to sulfuric acid play a major role in the observed potentiation of sulfur dioxide by certain particles.

D. CARCINOGENESIS

1. Carcinogens

The incidence of cancer and the insidious nature of the onset of malignant cell activity, together make essential the utmost effort to determine whether factors associated with air pollution can lead to occurrence of lung cancer in susceptible individuals or to increased susceptibility to cancer in the population at large. In various parts of the world, and especially in the United States, the relative mortality due to lung cancer has been increasing.[50] Furthermore, urban residents exhibit a greater liability to the development of lung cancer than do those living in rural areas,[51] and data from several investigations suggest that the epidemiological association between urban residence and lung cancer is of pathogenetic significance.[52-54] The association between lung cancer and cigarette smoking is too well documented to need further amplification here. There is, however, one feature common to both air pollution and cigarette smoking studies which should be emphasized. In no case, for the reasons explained in the introduction to this chapter, has a suspected carcinogen in fact been demonstrated experimentally to produce a lung tumor in man, although the epidemiological considerations to be developed in the next chapter may well show a significant association between the suspected material and cancer. Thus, substances such as the polynuclear aromatic hydrocarbons (of which benzo (a) pyrene, BaP, is the prime example) may or may not produce lung cancer in man. However, these hydrocarbons do increase tumor incidence in laboratory animals and in combination with specific particles or viral infection have produced malignancies. The risk taken with a potentially toxic substance whose effect may appear after a long period of latency should always be reduced to the minimum possible. The bulk of the ensuing discussion therefore is based on the hypothesis that carcinogens effective in animals may be significant in increasing human malignant tumor incidence, and the word "carcinogen" will be used without qualification.

A portion of the organic material present in the atmosphere as suspended particles (Table 1-2) may be carcinogenic, and carcinogenic materials have been identified in the atmosphere of virtually all large cities in which studies have been conducted. The incomplete combustion of organic matter is one of the major sources of such substances; the photochemical reaction products of aliphatic and aromatic constituents of gasoline in the presence of the atmospheric gases also possess some potency for generating tumors experimentally.

Chemical and physical studies of polluted urban air have been paralleled by investigations of carcinogens using skin-painting, subcutaneous injection, and inhalation techniques. The carcinogenicity (as measured by these techniques) of extracts of materials collected from air as well as such pollutant sources as chimney soots, road dusts, and vehicular exhausts has been established by many investigators. It should be noted first that, in many cases, use was made of mice (A-strain) particularly susceptible to tumor development; and second, that only two of the studies, those involving ozonized gasoline, used inhalation as a route of administration. The observed response in the case of A-strain mice inhaling ozonized gasoline was the relatively rapid development of multiple adenomas in the lung which, although tumors, are not malignant.[55]

More recently, squamous cell cancers of the lung were induced in C-57 black mice following infection with influenza virus and continuous exposure to an aerosol of ozonized gasoline. The tumors produced were histologically identical with those observed most frequently in man.[56]

Measurable concentrations of inorganic substances, such as metal dusts and asbestos, demonstrated to be occupationally associated with increased liability to lung cancer development, are also emitted into the atmosphere.[57-63] Additional laboratory experimentation is needed to verify such carcinogenic potential relative to interacting effects and respective ambient atmospheric concentrations.

2. Polynuclear Aromatic Hydrocarbons as Carcinogens in Polluted Atmospheres

Polynuclear aromatic hydrocarbons are commonly regarded with extreme suspicion as possible carcinogens, and much of the experimental

Table 10-2. BENZO(A)PYRENE CONCENTRATIONS IN SEVERAL URBAN AND NONURBAN AREAS.[64]

State	μg BaP/1000 m^3 air	
	Urban	Nonurban
Alabama	24	0.076
Indiana	39	1.8
Maryland	14	0.70
Missouri	54	0.025
North Carolina	39	0.25
Oregon	8	0.01
Pennsylvania	61	1.9
South Carolina	24	1.1

Table 10-3. BENZO(A)PYRENE AS A FRACTION OF THE TOTAL AROMATIC HYDROCARBON CONTENT OF SEVERAL URBAN ATMOSPHERES[65]

City	BaP fraction of total aromatic hydrocarbon, µg/g	
	Lot 19	Lot 20
Atlanta	1,800	1,900
Birmingham	2,300	3,400
Cincinnati	2,800	5,200
Detroit	3,800	4,500
Los Angeles	660	260
Nashville	5,900	4,900
New Orleans	2,100	1,600
Philadelphia	2,500	?
San Francisco	680	290

work performed has concerned these compounds. This section deals with the presence and stability of the hydrocarbons in the atmosphere, and with their elution from soots on which they may be adsorbed.

The concentration of benzo(a)pyrene (BaP) as a representative carcinogenic hydrocarbon in selected urban and nonurban areas within the United States is shown in Table 10-2;[64] in Table 10-3, the BaP content is given as a fraction of the total aromatic hydrocarbon content of air pollutants of nine American cities.[65]

The physical stability of aerosols in polluted atmospheres has already been described in general terms (Chapter 1). Chemically, BaP seems to be relatively stable and, even in the presence of a strongly oxidizing atmosphere, such as that found in photochemical smog characteristic of Los Angeles, the rate of disappearance of BaP is smaller than that of many other hydrocarbons. Table 10-4 shows the destruction under certain conditions of exposure of various polynuclear aromatic hydrocarbons present in air.[66]

Table 10-4. EFFECT OF VARIOUS CONDITIONS OF EXPOSURE ON THE DESTRUCTION OF SOME POLYNUCLEAR AROMATIC HYDROCARBONS[66]

Hydrocarbons	Percent destroyed						
	Pure unadsorbed					Adsorbed on soot	
	By air in dark		By air in light		By smog in light	By air in light	By smog
	Hours						
	24	48	24	48	1	48	1
Compound X	100		100		100	12	72
Anthanthrene	44	49	42	44		5	55
Phenanthrene	39	61	34	60			
Pyrene	24	43	20	42	83	1	58
Fluoranthene	17	20	16	24	59	4	59
3, 4 Benzpyrene	0	0	21	22	50	10	18
1, 2 Benzpyrene						7	51
1, 12 Benzperylene	0	0	0	0	27	0	67
Coronene	0	0	0	0	5		
Chrysene	0	0	11	0	15		

Polynuclear aromatic hydrocarbons may exist in the atmosphere adsorbed on carrier particles as well as in their free state. As mentioned in section C-2, the rate and extent of elution of adsorbates, either into the macrophages or onto the respiratory epithelium, are influenced by the size of the carrier particles inhaled. Polycyclic aromatic hydrocarbons cannot be readily eluted from soots of very small particle size; in fact, particles with an average diameter of less than 0.04 μm will remove these compounds from their immediate environment because of high surface adsorption. Particles above the 0.04-μm average size range will generally release adsorbed aromatic polycyclic hydrocarbons in the presence of appropriate solvents, which include plasma and cytoplasmic proteins. As particle size increases, release

Table 10-5. PERCENTAGE RECOVERY OF POLYNUCLEAR AROMATIC HYDROCARBONS FROM 0.5-μm SOOT PARTICLES AND FROM PLASMA AFTER INCUBATION WITH PLASMA FOR VARYING PERIODS[67]

Compound	Concentration, μg/mg Soot	Fraction	Incubation time, hours 1.5	16	96	192
			Percentage recovery			
Pyrene	3.4	Soot	9	34	9	6
		Plasma	81	61	50	61
		Total	90	95	59	67
Fluoranthrene	1.0	Soot	trace	present	0	present
		Plasma	100	present	present	present
		Total	100	—	—	—
Compound-X	2.8	Soot	0	trace	0	0
		Plasma	100	93	present	39
		Total	100	93	—	39
1,2-Benzpyrene	2.9	Soot	trace	17	0	0
		Plasma	52	41	21	21
		Total	52	58	21	21
3,4-Benzpyrene	1.7	Soot	18	41	6	18
		Plasma	82	59	23	18
		Total	100	100	29	36
1,12-Benzperylene	9.2	Soot	31	67	5	11
		Plasma	66	33	15	13
		Total	97	99	20	24
Anthanthrene	2.4	Soot	25	54	trace	13
		Plasma	54	33	17	13
		Total	79	87	17	20
Coronene	10.0	Soot	40	66	12	15
		Plasma	36	26	8	10
		Total	76	92	20	25

becomes more rapid and greater in extent. The elution of polycyclic aromatic hydrocarbons from 0.5-μm soot particles after incubation with plasma for various time intervals is shown in Table 10-5.[67] Asbestos mesothelioma may be the result of incorporation of and slow release of polycyclic aromatic hydrocarbons adsorbed on asbestos fibers as well as a primary effect of asbestos fibers themselves. Table 10-6 indicates

Table 10-6. AMOUNT OF POLYCYCLIC AROMATIC HYDROCARBONS[68]

Aromatic Hydrocarbon	µg/100g of Fiber	
	Crocidolite Oil	Amosite Oil
Pyrene	3	19
Fluoranthrene	present	38
Phenanthrene	present	65
Coronene	0.7	2.5
Anthracene	--	14
Anthanthrene	--	0.25
Benzo[a]phrene	1	5
Benzo[e]phrene	4	8
Benzo[g,h,i]perylene	1	3.2
Benzo[k]fluoranthrene	present	2.5

the polycyclic hydrocarbons extracted from crocidolite and amosite oils.[68]

In studies on skin carcinogenesis, Falk and Steiner,[69] using commercial carbon blacks, related the biological activity of adsorbed carcinogens to the size of the soot particle and the presence of natural eluting substances at the site of deposition. They advanced the principles of natural and conditional carcinogens, of solvent elution, and of adsorption, to explain some clinical and epidemiological observations of human skin and lung cancers, and of the role of preceding pathological lesions in predisposing to pulmonary tumors.

The interaction of carcinogens with other particulate agents has been studied toxicologically, utilizing laboratory animals. Experiments have shown that the addition of seemingly inert particulates to carcinogens results in the production of malignant neoplasms in the lung. Pylev[70] produced an appreciable incidence of lung cancers in rats by the intratracheal administration of 9,10-dimethyl-1,2 benzathracene (DMBA) incorporated with india ink in a 4-percent casein solution, whereas Gricute[71] was unsuccessful with the same material when it was suspended only in physiological saline. Saffiotti and coworkers[72,73] have produced a variety of malignant tumors in the lungs of hamsters by intratracheal instillation of saline suspensions of BaP ground together with hematite (Fe_2O_3) as a carrier dust in amounts equivalent to 3 mg of each chemical, once weekly for 15 injections. Not only was a high incidence of lung cancers produced but also these lu[illegible]cers mimicked all the various cell types seen in human cancers--tha[illegible]uamous cell carcinoma, anaplastic carcinoma, adenocarcinoma, and ev[illegible]cheal cancers. Dose-response effects were suggested, as were indications that a single high dose could induce cancers in this system.[73] According to Saffiotti et al.[74], the increased action for the carcinogen is thought to be brought about by its adherence to the fine inert particles which in turn carry it through the respiratory bronchioles and alveoli into the lung parenchyma. The carcinogens may then be eluted from the particles and spread diffusely to reach the target tissue. They infer that the hematite dust brings about a reduction in the speed in which BaP is removed from the respiratory tract. Shabad et al.[75] have reported that when india ink was included in the inoculum, BaP was eliminated more slowly from the lung. One must consider the possibility that the carrier material itself might be contributing some effect. Faulds and Stewart[76] reported increased incidence of carcinoma in the lungs of hematite miners. However, it is difficult to assess the exact role of iron in such a complicated exposure situation, since silica and other dusts are certain to be present. Bonser et al.[77] suggest that iron oxide may play a role in converting the fibrogenic effect of silica into a carcinogenic process. Haddow and Horning[78] have reported that the carcinogenic action of an iron-dextran complex cannot be entirely explained by the dextran content alone. Consequently, iron cannot be excluded as a possible contributing factor or cofactor in cancer production. Epstein et al.[79] have applied methods employing the injection of crude benzene-soluble extracts from atmospheric particles into suckling mice. The total dosages administered ranged from 5 to 55 mg and produced a high incidence of tumors in surviving mice as compared to controls. At 50 weeks post-inoculation, a variety of tumors was evident, the most significant being lymphomas, hepatomas, and multiple pulmonary adenomas. In mature mice injected subcutaneously with carcinogens, the tumors usually develop in the vicinity of the site of inoculation. However, in newborn mice, the tumors frequently develop at distal points such as the liver, lungs, or lymph nodes.

Kuschner[80] has demonstrated the interaction of a known carcinogen with the gaseous air pollutant, SO_2. Inhalation exposures of rats indicated that SO_2, alone, produced proliferative and metaplastic changes in the bronchial epithelium; BaP, alone, failed to cause the development of tumors; but the inhalation of BaP in the presence of SO_2 caused the development of bronchogenic squamous cell carcinoma. Hettche[81] conducted an epidemiological study on the incidence of human lung cancer in various areas of the Rhine Valley and concluded that excessive motor vehicle and diesel exhaust fumes coupled with polynuclear aromatic hydrocarbons casused a greater increase in lung carcinoma than any of the other gaseous or solid atmospheric pollutants.

3. Pathology of Carcinogensis

Rather little experimental evidence is available on the pathology of carcinogensis following exposure to air pollutants; much more experimentation has been carried out in relation to smoking and lung cancer. The results of the extensive work on the latter topic by Auerbach et al.[82] are reported briefly because of the possible similarities between carcinogenic mechanisms in cigarette smoking and air pollution. The authors describe a series of changes which are characterized by loss of cilia, by increase in the number of cell rows, and by the presence of atypical cells in the thickened epithelium. Their findings suggest a progression of changes from initial goblet cell hyperplasia to metaplasia, metaplasia with atypism, carcinoma *in situ*, and invasive cancer. Their data further support the epidemiological observation of a dose-response to cigarette smoke. Carnes[83] also observed a clear and pronounced association between the extent of epithelial change and exposure to pollutants, and emphasized the similarity of epithelial changes in man to "hyperplasia and other changes in the bronchial epithelium of mice." Indistinguishable histologic counterparts can be identified in both man and experimental animals.

The sequence of hyperplasia, metaplasia, metaplasia with atypical change, cancer *in situ*, and invasive cancer is likely to occur first at sites of impingement and particle retention. In experiments with animals, particle deposition and retention occur most readily in the more distal segments of the tracheobronchial tree, a result which appeared at first to be incompatible with early clinical and pathological

Table 10-7. DISTRIBUTION OF LUNG CANCER BY SITE OF ORIGIN[84]

Site of Origin	Percent of Cases
Main bronchus (including intermediate)	11
Lobar bronchus.	29
Segmental bronchus.	29
Segmental area (i.e., peripheral tumor)	31

observations that primary bronchial carcinomas originated chiefly in the main stem bronchi. Early clinical observations, however included a majority of cases in which tumor size was great and point of origin was difficult to ascertain, and later reports question the concept that most primary lung cancers are hilar or central in origin. For example, Table 10-7 shows the distribution of lung cancer by sites of origin as given by Kotin and Falk.[84]

E. SUMMARY

This chapter reviews toxicological studies of various types of particulate matter known to be present in ambient atmospheres. These studies are primarily concerned with the disciplinary areas of pathology, physiology, and carcinogensis. In addition, the mechanisms of the toxicological action of particulate matter are discussed and considerations concerning mixtures of irritant gases and particles are given proper emphasis. To date, studies using laboratory animals have been concerned, in the main, with concentrations of particles far in excess of ambient concentrations. Although direct extrapolations from the laboratory animal to man are impossible, animal experimentation is useful and necessary for rapidly defining the toxicological mechanisms of action and for determining the primary biological systems affected by a specific particulate material alone or in the presence of an irritant gas. Findings obtained from animal experimentation can be tested under community conditions with human populations through epidemiological studies. Particulate matter may exert a toxic effect through one or more of three mechanisms:

1. The particle may be intrinsically toxic due to its inherent chemical and/or physical characteristics,
2. The particle may interfere with one or more of the clearance mechanisms in the respiratory tract, and
3. The particle may act as a carrier of an adsorbed toxic substance.

The last of these mechanisms can lead to a "potentiating" effect in which particles containing an adsorbed toxic substance increase the physiological response to the adsorbed substance to a level above that which one would expect if the substance were present in the absence of the particle. Conversely, prior exposure of an animal to particulate matter can sometimes afford a degree of protection to subsequent exposure to irritant gases.

Particle size and dust load both contribute toward determining the toxicity of any specific chemical substances: reduction in particle size generally increases toxicity, while even an "inert" particle may elicit toxic responses when present in high enough concentrations. Finally, it has been clearly demonstrated that a number of substances (metal dusts, asbestos, polynuclear aromatic hydrocarbons, etc.) known to be carcinogenic in animals are present in polluted atmospheres.

When linked with urban-rural differences in lung cancer frequency revealed from epidemiologic investigations, this evidence indicates that such substances in urban polluted atmosphered may be potential carcinogens for the exposed human population.

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Chapter 11

EPIDEMIOLOGICAL APPRAISAL OF PARTICULATE MATTER

A. INTRODUCTION

Health effects produced by atmospheric particulate matter are discussed in this chapter in terms of epidemiological studies. Because particles tend to occur in the same kinds of polluted atmosphere as sulfur oxides, few epidemiological studies have been able to adequately differentiate the effects of two pollutants. It follows, therefore, that the studies presented in this chapter are frequently identical with those described in the companion document, Air Quality Criteria for Sulfur Oxides.

Epidemiological studies, as distinguished from toxicological and experimental studies, analyze the effects of pollution from ambient exposure on groups of people living in a community. Such studies have the advantage of examining illness where it occurs naturally, rather than in a laboratory, but carry the disadvantage of not being able to control precisely all factors of possible importance. Nevertheless, the preparation of air quality criteria must rest on epidemiological studies because of the very severe limitations of toxicological and experimental studies for this purpose.

In determining whether or not an association is causal, consideration must be given to several aspects of association which include strength, consistency, specificity, temporality, biological gradient, plausibility, coherence, and analogy.[1] A judgment of the value of an epidemiological study requires an understanding of these aspects. Many types of epidemiological evidence suggest that air pollution may exert considerable influence on health, as well as on the "satisfaction with life," of major segments of the world population.

Several health indices are described in section B-1; certain precautions which should be observed in the application of epidemiological methods to air quality criteria are suggested in section B-2. The studies themselves are listed in section C, according to the index employed.

B. APPLICATION OF EPIDEMIOLOGY TO AIR POLLUTION STUDIES

1. Indices

Various indices of health may be used for correlation with air pollution by the particulate matter. Among these are:

a. Mortality (greater than expected)

    (1) deaths from all causes

    (2) deaths from specific causes

    (3) deaths among the different age and sex groups

b. Morbidity

    (1) incidences of disease--chronic bronchitis, pulmonary emphysema, diffuse interstitial pneumonitis, cancer of respiratory tract, disease of remote organ (e.g., gastrointestinal, ophthalmic, and cardiovascular systems)

    (2) prevalence of diseases--same examples as for "incidences"

    (3) prevalence of respiratory symptoms (e.g., changes in quality and/or quantity of sputum produced)

    (4) exacerbation of diseases--rhinorrhea, asthma, tracheobronchitis, and chronic illness

    (5) enhancement of infections--pneumonia, sinusitis, otitis, mastoiditis

(6) changes in clinical conditions (e.g., for bronchitic patients)

c. Changes In Various Aspects of Lung Function

(1) ventilatory function--decrease in peak flow rate, decrease in spirometric volumes, impairment of flow-volume relationship, and increased airway resistance

(2) blood/gas distribution--impairment of lung-gas distribution

(3) blood/gas exchange--impairment of pulmonary blood-gas exchange

(4) increased work of breathing

Before considering individual epidemiological studies, some discussion is necessary concerning the problem of relating air pollution and its effects through community health indices. This discussion is in the form of a series of cautions pertaining to the interpretation of data.

2. Cautions

In the first place, as discussed in Chapter 1, methods of measurement vary from country to country and place to place. Results from the high-volume sampler and results from the spot samples are very dissimilar. Unless measurement procedures and equipment have been standardized and common calibration methods are used, results of studies undertaken at one location cannot be considered applicable at another.

Secondly, particle size plays an important part in the selection of the measuring technique as it does in determining the site and effectiveness of deposition in the respiratory tract. (See Chapter 9.) However, size distribution of particles has nearly always been ignored in field studies.

Thirdly, pollution and health indices are not always measured over the same time periods. It is to be hoped that the pollution levels cited bear some relation to those extant during the time when the chronic disease states were developing. Further, acute health effects require frequent short-term pollution measurements to enhance detection, while long-term chronic processes may be adequately related to long sampling intervals. In many epidemiological studies the possible role of cigarette smoking has not been considered. It is expected that future epidemiological studies involving adults will routinely collect data on smoking habits of the study group. Other factors are significantly related to respiratory disease. These include occupational and other past exposures; infections, past and present; and allergy and heredity.

Few or no epidemiological studies have been possible where the pollution challenge has been limited to particulate matter, unaccompanied by significant amounts of other pollutant substances. Indeed, most of the available conclusions link particle concentrations with those of concurrently measured sulfur dioxide; some studies attempt statistical separation of the culpability of one factor from the other in the effects cited.

In seeking the possible effects on populations resident in areas of differing air pollution, factors such as smoking, type and conditions of employment, ethnic group, and mobility in response to experienced irritation or disease have sometimes been considered. There has, however, been a minimum of attention paid to the indoor or domestic environments and their potential contribution. Measurement of such indoor exposures might be difficult, but omission of the information could well modify the appraisal of the importance of particulate pollution.

Toxicological studies indicate a specific potential of some particulate materials to produce human responses. The levels used in toxicological studies are far higher than those found in the communities in the epidemiological studies under review. Thus the actual responsibility of particles for the community responses is uncertain, and it is sometimes necessary to invoke additional concepts--for example, the idea of synergism with other known or unknown ambient pollutants, or the idea that particulate matter is but an index of availability of some other substance(s) which are fully responsible for the effects reported.

Over a short period of time, mortality fluctuates considerably, and only a systematic, long-term approach will allow a valid determination of the real role of air pollution. Cassell et al.[2] have reviewed the problem of detecting peaks in mortality and relating them to any single variable. The danger with episodic studies is that a short-term fluctuation in the death rate, when picked to coincide with an air pollution incident, may appear to be causally related; in the long term, however, numerous other unassociated peaks are found in both the death rates and the air pollution levels.

Specific substances encountered as respirable particles (beryllium, asbestos, arsenic, vanadium, lead, airborne pathogens, etc.) and associated with disease entities are not assessed in this chapter. They deserve consideration, however, as exemplars of the modes of response of which the human is capable. These responses may delay or confound recognition of the importance of "particulate matter" inhalation. The demonstration of the importance of aerodynamic behavior rather than of mass or particle dimension for both impingement and retention of asbestos is striking. Long-delayed effects from relatively brief community exposures to asbestos and beryllium are significant (see Chapter 10). Furthermore, penetration of "poorly cleared" particles of asbestos to remote body sites suggests complex body transport mechanisms, differing organ or tissue sensitivities, and the need for evaluation of as yet untested relationships between disease entities and respirable offenders. These examples confer some sense of urgency to establishing the relationships of human disease and dysfunction to air pollution by "general particulate matter."

The concept of "susceptible population" demands consideration. Human responses to toxicants, and to community air pollution, show wide variations, which contribute in no small way to the difficulty in assessing in a general manner the effects of pollutants. Since air quality criteria, unless otherwise specified, must consider the total population rather than just major segments of it, studies must consider especially the impact of air pollution on the "most sensitive" responders. Many factors seem to enhance susceptibility or sensitivity to air pollution. These include being at the extremes of age (i.e., infants and the very old); having preexisting chronic respiratory disease (e.g., pulmonary emphysema or chronic bronchitis); having preexisting cardiovascular disease (functional capacity not defined); regularly smoking cigarettes; or living in overcrowded or depressed socioeconomic strata. Some of these factors have been singled out for attention in the references to be cited, and the level of pollutant said to have an "effect" may take cognizance of such special sensitivity.

The effects discussed are related insofar as possible to specific pollution over specific time intervals; it must be emphasized that lower pollution values by no means imply a "no effect" level of the pollutants.

C. INDICES OF HUMAN RESPONSE: THE EPIDEMIOLOGICAL STUDIES

1. Acute Episodes

a. Mortality

In conurbations, such as London, New York, Chicago, and Detroit, it has been possible to observe deviations from the moving averages of deaths during various seasons and to relate such deviations to the coincident period levels of air pollutants.[3]

In writing about the Meuse Valley episode in 1936, Firket stated that if there were a similar phenomenon in London, some 3,200 deaths might occur.[4,5] Unfortunately, he was quite accurate in his estimate since, in December 1952, the world's most disastrous smog incident occurred in London, coincident with about 4,000 excess deaths throughout the Greater London area. Marked increases were noted in both respiratory and cardiovascular deaths (and for almost every cause except traffic accidents; presumably the smog was too thick for people to drive). Since some of the diseases such as lung cancer and tuberculosis were obviously existent before the pollution episode, much of the effect of the smog was to hasten the death of people who were already ill. Detailed investigations were made of 1,280 post-mortem reports of persons who had died before, during, or shortly after the episode. No fatalities were found which could not have been explained by previous respiratory or cardiovascular lesions. In this episode as in others, the elderly and persons with preexisting pulmonary and cardiac disease were most susceptible.

A number of investigations have analyzed and compared the various London episodes. The report by Brasser et al.[6] appears to cover all the episodes and to present a detailed analysis of each of these episodes, pointing out the importance of the duration of the maximum values of pollution concentration. More recently, Joosting[7] has examined the relationship between the duration of maximum values of sulfur dioxide and smoke during air pollution episodes, as well as the differential relationship between sulfur dioxide and smoke levels and the resulting mortality.

Gore and Shaddick[8] and Burgess and Shaddick[9] reviewed acute "fog" episodes which occurred in London in 1954, 1955, 1956 (January and December), and 1957 (December), in terms of excess mortality above a moving average, related to the mean of daily readings at seven stations for smoke and SO_2. Figure 11-1 shows mortality figures for the January 1956 and December 1957 episodes.[9] Somewhat differing patterns of onset of mortality rise, of age of population suffering most heavily, of deaths related to bronchitis and to other respiratory diseases, and of total deaths were noted in these acute episodes. Common to them, however, were elevations in the mean daily levels of SO_2 and smoke, measured at seven different stations, from 2 to 4 times the winter average levels; pollutant levels were estimated at 2,000 $\mu g/m^3$ black suspended matter together with 1,145 $\mu g/m^3$ (0.4 ppm) SO_2 (representing all acidic gases). Deaths ascribed to bronchitis were materially affected, but deaths due to other causes also increased. Deaths appeared to begin to increase before the onset of the episodes; during the episodes, of course, they increased substantially. Scott[10] observed

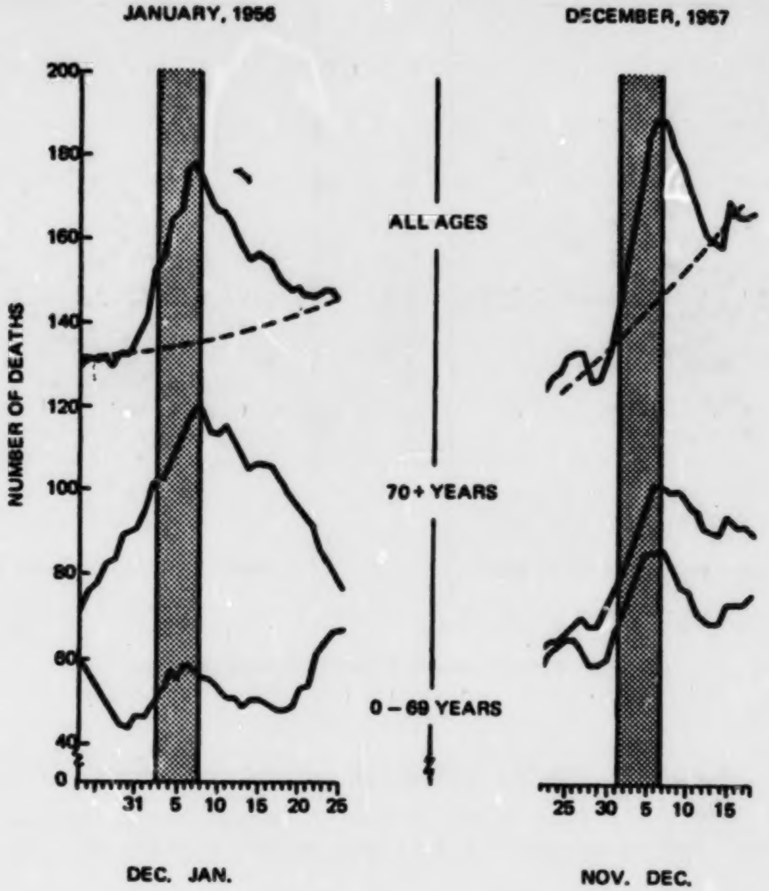

Figure 11-1. Mortality figures for the January 1956 and December 1957 smog "episodes" in London.[9] (The figure shows the increase in numbers of deaths during smog "episodes" --shaded periods--especially in the older age group.)

a similar relationship, for similar periods of "fog," with corresponding polutant levels. There was a sharp impact on the elderly, and bronchitis, as a cause of death, showed the greatest proportionate increase.

Martin and Bradley[11] correlated daily mortality (all causes) and daily brochitis mortality with mean daily black suspended matter (see

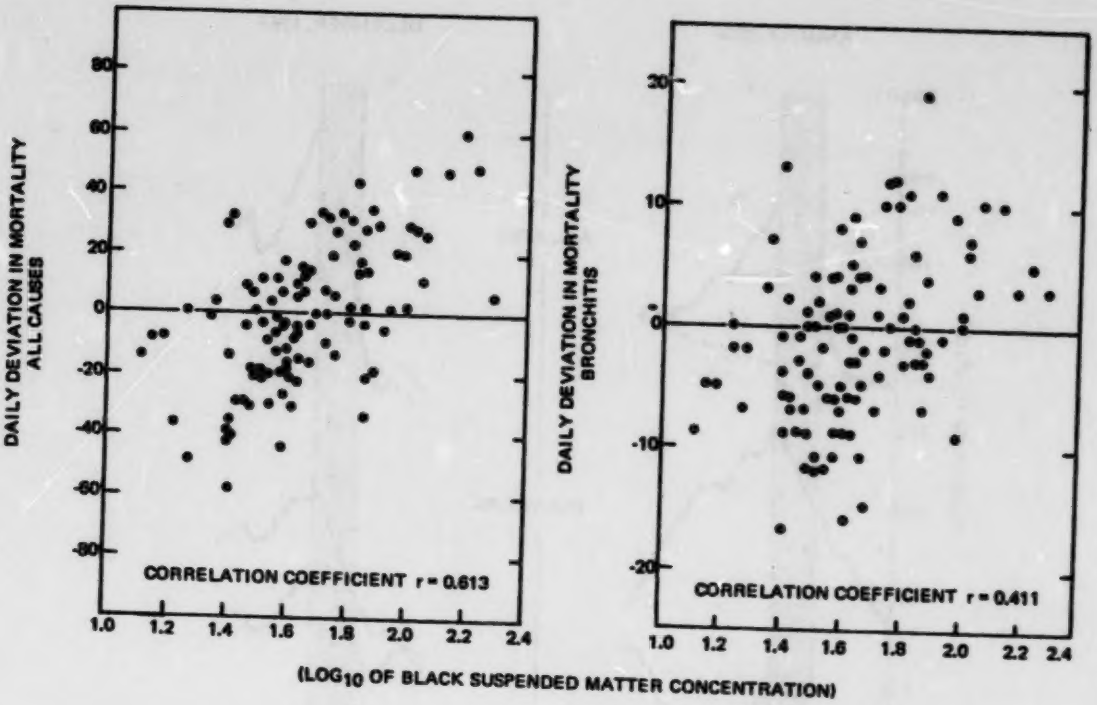

Figure 11-2. Mortality and air pollution in greater London during the winter of 1958-1959.[11] (The figure shows increased mortality due to "all causes" and to bronchitis during a period of high pollution.)

Figure 11-2) measured at seven locations in London for the winter of 1958-1959, and also found a significant positive association between mean daily sulfur dioxide concentration and deaths (all causes). Bronchitic deaths showed a lower correlation with the pollution level, and the authors suggested the need for consideration of effects of air pollution on patients with cardiovascular disease. In addition, excess deaths have been related to increases (on the day preceding death) of mean daily black suspended matter by more than 200 μg/m^3, and to rises in mean daily SO_2 of more than about 75 μg/m^3 (2.5 pphm). In a later paper,[12] data are shown to suggest increases in mortality, from all causes, and in respiratory and cardiac morbidity, associated with levels of smoke about 1,000 μg/m^3 and with SO_2 concentrations of 715 μg/m^3 (25 pphm). This "effect is properly related to abrupt rises in the concentrations of smoke and/or SO_2 with perhaps a continuum of effects at lower levels. Since these measurements were obtained at a single point in central London, it should be presumed that a relatively wide range of values around these levels actually contributed to the mortality statistics which were correlated. A reanalysis by Lawther[13] of these mortality studies indicates that the mortality increase is apparent at about 750 μg/m^3 for smoke and about 715 μg/m^3 (0.25 ppm) for SO_2. Joosting[7] states that the maximum sulfur dioxide concentration above which significant correlation occurs with death and disease is 400 to 500 μg/m^3 (0.15 to 0.19 ppm)* when there is a high soot content.

The Dutch report on sulfur dioxide,[6] which discusses in detail seven air pollution episodes in London, states that in the December 1956 episode, 400 excess deaths, or 25 percent above expected, were observed in Greater London at maximum 24-hour levels of 1,200 μg/m^3 for smoke and 1,100 μg/m^3 (0.41 ppm)* for SO_2. The report also notes that in January 1959, 200 excess deaths were observed in Greater London, or 10 percent above expected mortality, at a level of 1,200 μg/m^3 for smoke and 800 μg/m^3 (0.30 ppm)* for SO_2. The episodes all took place during winter; cold weather seems to have been an important feature of London's air pollution mortality.

In Martin's review[12] in 1964 of daily mortality in London during the winters of 1958-1959 and 1959-1960, he concluded: "From the data it would be difficult to fix any threshold value below which levels of air pollution might be regarded as safe." However, his review included data with smoke concentrations ranging upward from 500 $\mu g/m^3$ and accompanied by sulfur dioxide concentrations of about 400 $\mu g/m^3$ (0.14 ppm) and above.

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*SO_2 is converted from parts per million to micrograms per cubic meter in the Dutch report by using the equivalency 2,700 μg/m^3 = 1 ppm; this report uses 2,860 μg/m^3 = 1 ppm.

As a result of smoke control regulations, the particle content of London air has steadily decreased since the 1950's, but the sulfur dioxide concentrations have not decreased proportionately. At the same measuring sites as in 1952, sulfur dioxide was actually slightly higher in the 1962 episode than in that of 1952, but smoke levels were considerably lower. Also, as Brasser et al.[6] have noted, there was only 1 day of maximum pollution values in 1962 as contrasted with the 2 days of maximum pollution in December 1952. Although the smoke levels appear to be better related to the amount of excess mortality, other factors must be considered as possibly also reducing the number of deaths. Since 1952, a great deal of publicity has been given to the harmful effects of smog, and more susceptible individuals have been encouraged to use masks and filters and to stay indoors. In addition, when episodes come close together, the excess mortality in the early episodes reduces the number of susceptible individuals who experience the later episodes. An effect as large as that seen in the first incident would not, therefore, be expected.

The number of deaths in New York City was reviewed for excess mortality in relation to the air pollution episode of November 1953, by Greenburg et al.[14] Excess deaths were related to elevations of concentrations of sulfur dioxide and suspended particles. Average daily suspended particulate matter measured in Central Park was in excess of 5 coh units, while the SO_2 rose from the New York City average ranges of 430 to 570 μg/m^3 (0.15 to 0.20 ppm) to a maximum level of 2,460 μg/m^3 (0.86 ppm). For this episode, there was a "lag effect," and distribution of excess deaths among all age groups was noted. The number of deaths, although not showing the marked rise seen in some of the London episodes, was above average for comparable periods in other years during and immediately after the incident. For the November 15 to 24, 1953, period, the average number of deaths per day was 244, whereas during the 3 years preceding and following 1953, the average was 224 deaths per day for the same calendar period.

A later episode (1962) was studied, but Greenburg et al.[15] did not discern an excess mortality. However, McCarroll and Bradley,[16] reviewing episodes in New York City in November and December of 1962, January and February of 1963, and February and March of 1964, compared 24-hour average levels of various pollutants with New York City mortality figures, employing daily deviations from a 15-day moving average; the pollution measurements were obtained at a single station in lower Manhattan and fluctuations in the values at this station were known to correlate well with those at another station 10.5 km away. Excess deaths on December 1, 1962, followed a daily average SO_2 concentration of 2,060 μg/m^3 (0.72 ppm) and smoke shade in excess of 6 coh units, during a period of atmospheric inversion and low windspeed. The increased death rates were shared by the 45-to-64 age group, as well as by the age group over 65. In a later episode (January 7, 1963) associated with an SO_2 concentration of 1,715 μg/m^3 (0.6 ppm) and a smoke shade of 6 coh units, there was a peak of death rate apparently superimposed upon an elevated death rate average due to the presence of influenza virus in the community. Two further episodes[16,17] also suggest the relationship of excess deaths to abrupt rises in both SO_2 and suspended particles at times of air stagnation.

An example of thyinherent danger of relating mortality peaks to air pollution is shown by Leonard et al.[18] in the Dublin studies. During the war and postwar years of 1941 to 1947, peat rather than coal, was burned as the main fuel, and air pollution (as measured by particle concentrations) was markedly decreased. Sulfur dioxide levels varied in a manner similar to those of suspended particulate matter. The winter peaks in death rate, however, were unaffected, and thus do not seem to be related to air pollution. When coal again became available in 1948, the air pollution levels rose with no apparent effect on the death rate (see Figure 11-3). Unfortunately, it is not possible to assess the effects on these data of changing medical practices and the advent of antibiotics for use in treating respiratory diseases.

In Detroit[19] rises in infant mortality and deaths of cancer patients, occurring over a 3-day period, accompanied a rise in the 3-day mean suspended particulate matter for the same period above 200 $\mu g/m^3$ and an SO_2 maximum of 2,860 $\mu g/m^3$ (1.0 ppm), September 1952.

In Osaka, Japan, Watanabe[20] reported on excess deaths in a December 1962 smog episode. There were 60 excess deaths during a period of mean daily concentrations of suspended matter greater than 1,000 $\mu g/m^3$, with accompanying SO_2 concentrations greater than 285 $\mu g/m^3$ (0.1 ppm); the measurements were made at a single station in the central commercial area of the city.

When a marked increase in air pollution is associated with a sudden dramatic rise in the death rate or illness rate lasting for a

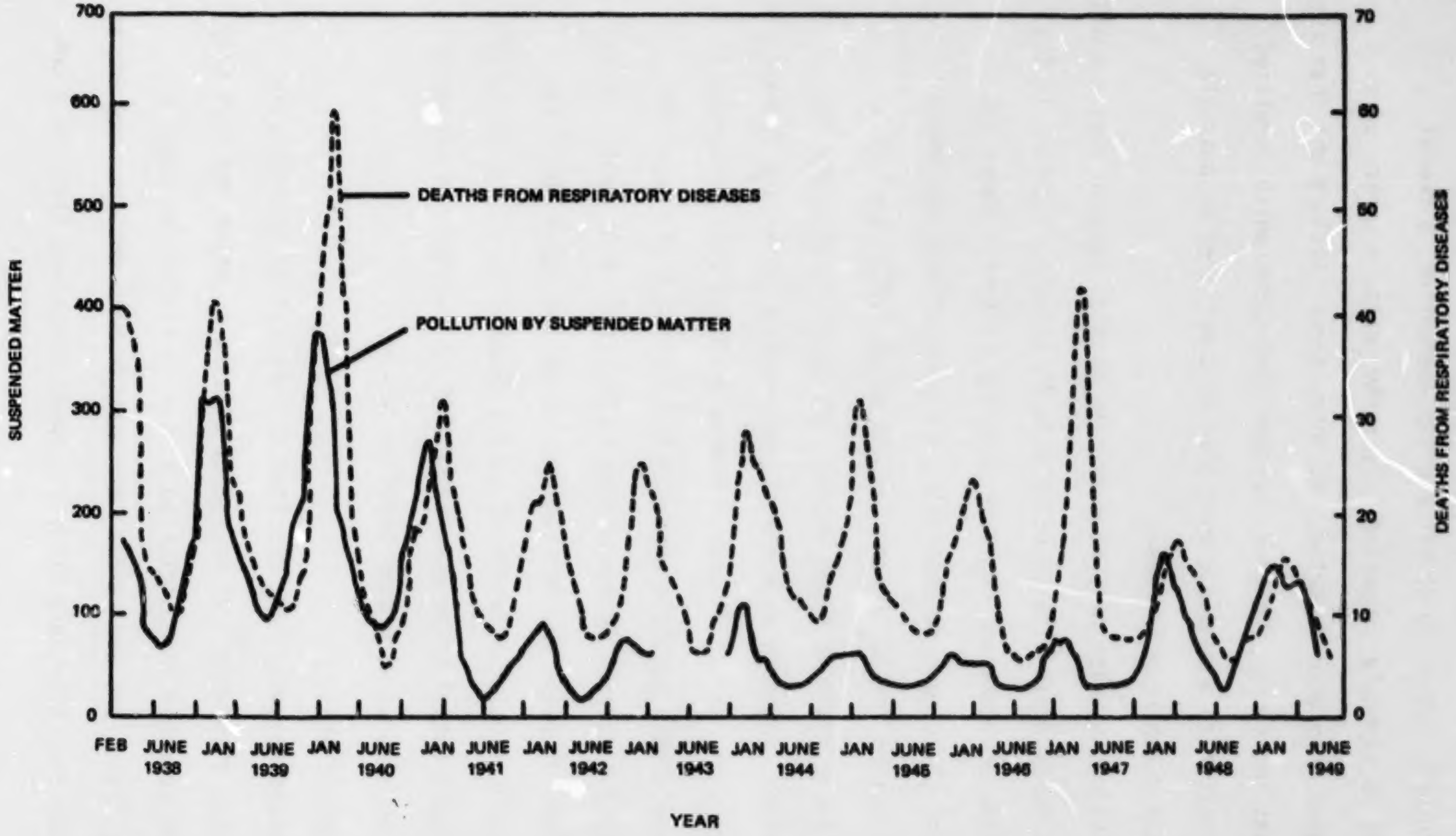

FIGURE 11-3. Death rates and air pollution levels in Dublin, Ireland, for 1938-1939.[18]

few days and both return to normal shortly thereafter, a causal relationship is strongly suggested. Over the years, a number of such acute episodes have been reported, and there seems little doubt that air pollution was the cause. However, weather conditions which permitted the air pollution to accumulate must also be considered as possible causes of the death rate increase.

The British studies presented in this section suggest that a small rise in the daily death rate is detectable in large populations if the concentrations of smoke and SO_2 rise abruptly to levels above 750 and 715 $\mu g/m^3$ (0.25 ppm) respectively; the same effects are noted in American cities for SO_2 concentrations above 1,700 $\mu g/m^3$ (0.6 ppm) and a smoke shade of 6 coh units. The major targets are the aged population, patients with chronic obstructive pulmonary disease, and patients with cardiac disease. A more distinct rise in deaths is noted generally when particle concentrations reach 1,200 $\mu g/m^3$ for 1 day and sulfur dioxide levels exceed 1,000 $\mu g/m^3$ (0.35 ppm). Daily concentrations of suspended particulates exceeding 2,000 $\mu g/m^3$ for 1 day in conjunction with levels of sulfur dioxide in excess of 1,500 $\mu g/m^3$ (0.5 ppm) appear to be associated with an increase in the death rate of 20 percent or more over baseline levels.

b. Morbidity

The acute episodes have resulted in substantial increases in illness. Thus a survery[21] of emergency clinics at major New York City hospitals in November 1953 indicated a rise in visits for upper respiratory infections and cardiac diseases in both children and adults in all of the four hospitals studied. Smoke shade was close to 3 coh units, and sulfur dioxide concentrations had not yet exceeded 715 $\mu g/m^3$ (0.25 ppm) when hospital admissions clearly rose.[14]

Again, the number of emergency clinic visits for bronchitis and asthma at seven large New York City hospitals was examined during the Thanksgiving 1966 air pollution episode.[22] There was a rise in the number of such visits on the third day of the episode, among patients age 45 and over, at three of the seven hospitals investigated. Unfortunately, the Thanksgiving holiday greatly complicated evaluation of the emergency clinic visits over the holiday period.[17]

In the investigation of the London episode of December 1952, information on illness was collected from as many sources as possible including sickness claims, applications for hospital admission, pneumonia notifications, and records of physicians. The analysis demonstrated a real and important increase in morbidity, though there was some indication that the increase in illness was not as large proportionately as the increase in deaths, and a marked rise did not occur in the early days of the episode. In a number of other severe London episodes, the increase in morbidity put a considerable strain on the health services.

These episodes reflect results which fall into Level IV of the World Health Organization's "guides to air quality."[23] These guides cover sets of concentrations and exposure times at which specified types of effects are noted or at which no effect is noted. Level IV includes "concentrations and exposure times at and above which there is likely to be acute illness or death in susceptible groups of the population."

2. Chronic (Long-Term) Air Pollution

Kurland[24] has pointed out that air pollution episodes represent, by definition, massive, overwhelming, and unusual exposure, and thus the most significant pathological effects. However, the ever-present lower concentrations of pollutants cannot be ignored.

a. Day-to-Day Variations in Mortality and Morbidity

In a systematic approach to analyzing respiratory and cardiac morbidity daily in London, Martin[12] examined deviations in morbidity from a 15-day moving average in London during the winters of 1958-1959 and 1959-1960. Both smoke and sulfur dioxide concentrations appear to be about equally related to morbidity rates, and a definite excess in morbidity seemed to occur with high pollution, as it did for mortality, though there was a somewhat greater degree of irregularity.

An approach similar to Martin's,[12] but limited to observations on mortality, was used by McCarroll and Bradley[16] in New York City. Covering a 3-year period (1962-1964) they examined a number of peaks in New York City mortality associated with periods of high air pollution. There are examples given where sulfur dioxide and smoke shade appear to be related to mortality. The authors present other episodes, however, where the relationship to air pollution is not nearly as clear, although the death rate fluctuates to even higher peaks. Reference to this analysis has already been made in our discussion of the data on episodes in section B-1.

McCarroll et al.[22] studied residents of a New York City housing project, using weekly questionnaires. Exact levels of air pollutants which could be used for establishment of air quality criteria were not given; however, the data indicate that sulfur dioxide rather than particulate matter was associated with eye irritation. Symptoms of cough were also shown to be related to air pollution but were not well differentiated between association with particles and sulfur dioxide. The particular time-series analysis used with these data is not well known, and the biases inherent in its use have not been fully determined.

b. Geographical Variations in Mortality

(1) Studies Based on Available Data. Mortality and morbidity statistics each have advantages as well as disadvantages. Records of illness should be more fruitful in defining subtle effects, since illness precedes death and since all illness does not result in death. Mortality statistics are collected in every country and are available for quick tabulation. Unfortunately, the quality of mortality statistics varies. One of the problems is that with the present system of tabulating mortality data, a single cause of death must be selected and coded, even though more than one cause may be involved in the death. The single cause of death designated (e.g., specific chronic respiratory disease) depends largely on the judgment of the attending physician and has little, if any, relation to epidemiological use. While contributing causes of death appear on the death certificate, they are not reflected in summary tabulations. The coding of only the "underlying" cause of death minimizes the importance of such diseases as emphysema which often appear on the death certificate as contributory or associated causes of death.[25]

Almost all studies of the effects of long-term exposure to air pollution on death rates compare the rate in one area with that in another. Mortality as well as morbidity studies are hampered by the possibility of differences, such as social class, occupation, age, and sex composition of the population, and cigarette smoking, other than air pollution existing between the areas. Assuming that almost all deaths are recorded and tabulated, comparison of total mortality rates (i.e., deaths from all causes) obviates the bias of diagnostic selection, but does not lessen the chances of other associated factors having caused the difference.

Buck and Brown[26] reported in 1964 the relation of standardized mortality ratios for the 5-year period 1955-1959 to four variables: daily smoke and SO_2 concentrations for March 1962 (presumed representative of the study period), population density in 1961, and a social index of 1951. The studies involved populations in 214 areas of the United Kingdom (19 London boroughs, 49 county boroughs, 70 boroughs, 61 urban districts, and 15 rural districts). The statistical studies indicate that bronchitis mortality had a significant positive association with both the smoke and the SO_2 concentrations encountered in these residential areas, and also with social indexes. The standardized mortality rates for lung cancer were not, in general, significantly associated with smoke or SO_2 concentrations in the residential areas. Examination of the tables given by Buck and Brown[26] suggests that the excess of bronchitis mortality occurred for classes of areas where the average daily smoke and SO_2 concentrations both exceeded 200 $\mu g/m^3$. Although smoking habits were reviewed and were apparently uniform from area to area, occupational and domestic indoor environmental exposures were not considered. The pollutant values selected for the correlations do not cover the same time period as the mortality figures.

A series of papers by Stocks[27-29,31] and by Stocks and Davies[30] related atmospheric pollution in urban and rural localities with mortality due to lung cancer, bronchitis, and pneumonia. Standardized mortality ratios apparently refer to the period 1950 to 1953 for bronchitis and pneumonia and to the period 1950 to 1954 for lung cancer. Lung cancer mortality was found to be strongly correlated with smoke density in the atmosphere for 26 areas of northern England and Wales, for 45 districts of Lancaster and Yorkshire, and for 30 county boroughs. (Similar but weaker correlations were observed within Greater London.) Further, social differneces in the populations concerned, only partially explain this correlation. Bronchitis and pneumonia for males and bronchitis for females similarly showed strong correlations with smoke density in the atmosphere. Cancers of the stomach and intestine in the county boroughs were also related significantly to smoke concentrations, and other relationships are described for various areas of the country. It is, difficult however, to extract specific quantitative levels at which smoke or the other pollutants studied produce effects. Papers[30,31] describe the statistical elimination applied to develop significant correlations of 13 trace elements, identified by spectrographic analyses, with mortality rates. Reanalysis of the data by Anderson[32] confirmed the importance of smoke levels, as well as social and population parameters, to mortality from lung cancer; vanadium is also identified as an important independent contributor to lung cancer, female cancer, and pneumonia mortalities.

In summary, the results of this analysis of long-term mortality indicate effects which would coincide with Level III of the World Health Organization's "guides to air quality."[23] Level III is defined as "concentrations and exposure times at and above which there is likely to be impairment of vital physiologic functions or changes that may lead to chronic diseases or shortening of life."

(2) Special Studies Involving the Collection of New Data. In 1964, also, Wicken and Buck[33] reported on a study of bronchitis and lung cancer mortality in six areas of northeast England--one in Eston, another in Stockton, and four in rural districts. The deaths covered the period 1952 to 1962. The survey of decedents with cause of death from bronchitis or lung cancer was matched against the survey of decedents with cause of death from nonrespiratory disease controlled for age and sex; the basis for the diagnostic classifications was not stated in the report. Personal interviews were carried out with next of kin. Personal interviews of a random sample of households were also conducted to obtain sex, age, smoking habits, and occupation of the population at risk, the living population. The survey of decedents was carried out between January and October 1963; the survey of the living population was carried out between December 1963 and March 1964. Smoke and sulfur dioxide concentrations were measured in the Eston urban district. One year's aerometric data was obtained. The study was excellent in principle though, unfortunately, sulfur dioxide and particulate values were available only for the Eston urban district.

Eston, itself, as a substudy, was subdivided into north Eston and south Eston. North Eston contains or lies near heavy industrial plants, whereas south Eston is a residential area. During the period May 1963 to April 1964, mean weekly observations of the sulfur dioxide and smoke concentrations were carried out in two sites in north Eston and one station in south Eston. Smoke values were 160 and 80 $\mu g/m^3$ for north and south Eston respectively. The sulfur dioxide value in north Eston on the yearly average was 115 $\mu g/m^3$ (0.040 ppm) and for south Eston it was 74 $\mu g/m^3$ (0.026 ppm). Adjustments were made for differences in age composition, smoking habits, and social class, and these were insufficient to explain the differences in lung cancer and bronchitis mortality rates between the two localities. Occupational exposure to pollution was then taken into account in the analysis. The conclusion was that there is an association between the degree of air pollution and the incidence of lung cancer and bronchitis mortality in the two areas of the Eston urban district. Though both sulfur dioxide and smoke values and concentrations are furnished in the report and the effects apparently cannot be separated, Brasser et al.[6] apparently have used the sulfur dioxide concentration as the more relevant measure of this study.

The community of Salford was classified into three pollution areas by Burn and Pemberton,[34] according to Table 11-1. Five sampling stations in the area were employed. Despite the closeness of the ranges of values, high rates of bronchitis mortality, of lung cancer mortality, and of deaths from all causes were observed in the higher rather than the lower pollution wards. It appears (see section C-2-d) that there was also an increased rate of bronchitis morbidity in the highly polluted wards.

Winkelstein and his coworkers[35-37] analyzed pollution effects in a group of studies made in the Buffalo, New York, area. In July 1961, 21 air-sampling stations were set up in and around the city of Buffalo, and daily values for suspended particles, dustfall, and oxides of sulfur were taken until June 1963. Suspended particle levels were used as

Table 11-1. POLLUTION LEVELS IN SALFORD (SEASONAL DAILY AVERAGES)[34]

Pollution area classification	Smoke $\mu g/m^3$		SO_2 $\mu g/m^3$ (ppm in parentheses)		Deaths $\frac{\text{observed}}{\text{expected}} \times 100$		
	Winter	Summer	Winter	Summer	All causes	Bronchitis	Lung cancer
High	680	270	715 (0.25)	255 (0.09)	106	128	124
Intermediate	490	170	460 (0.16)	200 (0.07)	100	97	84
Low	450	170	340 (0.12)	145 (0.05)	90	52	79

Note: The data in the original report show SO_2 concentrations in parts per hundred million (pphm).

the index of air pollution. On the basis of the measurements, the study area was divided into four contiguous areas according to their levels of air pollution. Level 1 was less than 80 $\mu g/m^3$ (2-year geometric mean); level 2, 80 to 100 $\mu g/m^3$; level 3, 100 to 135 $\mu g/m^3$; and level 4, greater than 135 $\mu g/m^3$. Each area was also divided into five economic groupings. Mortality figures for the area were taken from the period 1959 to 1961, with the 1960 census providing the population basis.

Annual death rates per 100,000 population for (1) all causes of death; (2) chronic respiratory disease; (3) malignant neoplasms of the bronchus, trachea, and lung; and (4) gastric carcinoma were related to the four areas of air pollution itensity and to the socioeconomic levels of the deceased.

Among white males between 50 and 69 years of age, the death rate from all causes was 20 per 1,000 in the area with the least suspended particulates (less than 80 $\mu g/m^3$). It was 20 percent higher in the next most polluted area (between 80 $\mu g/m^3$ and 100 $\mu g/m^3$ and twice as high in the most polluted area (more than 135 $\mu g/m^3$). (See Table 11-2 and Figure 11-4.[35])

Table 11-2. AVERAGE ANNUAL DEATH RATES PER 1,000 POPULATION FROM ALL CAUSES ACCORDING TO ECONOMIC AND PARTICULATE LEVELS, AND AGE: WHITE MALES, 50-69 YEARS OF AGE, BUFFALO AND ENVIRONS 1950-1961[35]

Economic Level	Particulate Level				
	1 (Low)	2	3	4 (High)	Total
1 (low)	—	36	41	52	43
2	24	27	30	36	29
3	—	24	26	33	25
4	20	22	27	—	22
5 (high)	17	21	20	—	19
Total	20	24	31	40	26

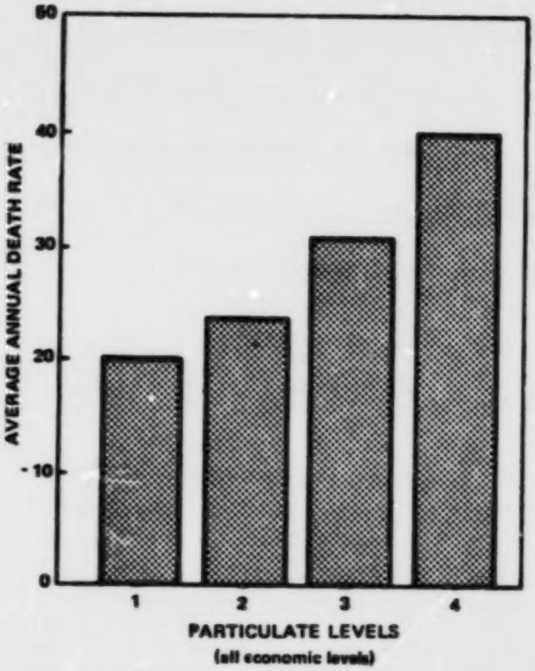

Figure 11-4. Average annual death rate from all causes. (The graph shows the death rate per 1,000 population for white males between 50 and 69 years of age for four levels of particulate matter, Buffalo and environs, 1959-1961.)[35]

Among white males between 50 and 69 years of age, the death rate from chronic respiratory disease was 42 per 100,000 population for those living in the area with the least suspended particulates, 40 percent higher for those living in the next most polluted area, and about 3 times as high for those living in the most polluted area. (See Table 11-3 and Figure 11-5[35].) Suspended particle concentrations did not appear related to deaths from cancer of the bronchus, trachea, or lung. However, positive correlations were established with suspended particle concentrations and total mortality, and mortality from chronic respiratory disease, regardless of the economic level of the deceased. Moreover, a positive correlation was established between concentrations of suspended particles and gastric cancer that appeared to be independent of economic status. It should be noted, however, that this study did not take into account smoking habits.

Table 11-3. AVERAGE ANNUAL DEATH RATES PER 100,000 POPULATION FROM CHRONIC RESPIRATORY DISEASE ACCORDING TO ECONOMIC AND PARTICULATE LEVELS, AND AGE: WHITE MALES, 50-69 YEARS OF AGE, BUFFALO AND ENVIRONS, 1959-1961[35]

Economic Level	Particulate Level				
	1 (Low)	2	3	4 (High)	Total
1 (low)	—	0[a]	126	188	133
2	64	75	96	105	84
3	—	65	51	103[a]	64
4	35	47	114	—	52
5 (high)	42	63	0[a]	—	50
Total	44	62	94	129	72

[a] Rate based on less than five deaths.

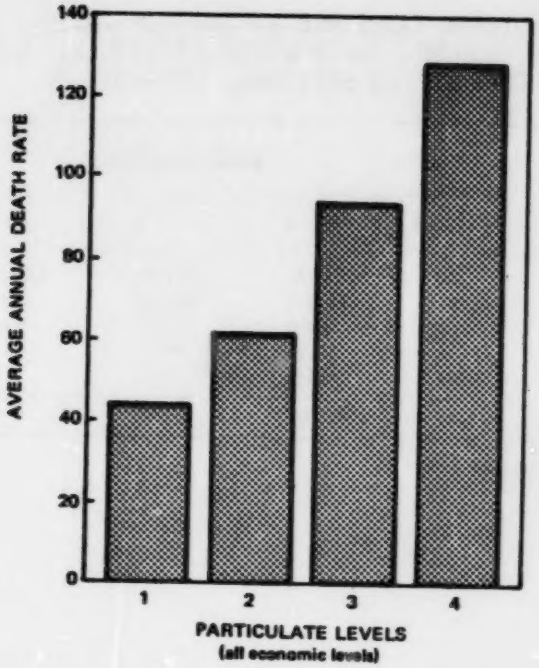

Figure 11-5. Average annual death rate from asthma, bronchitis, or emphysema indicated on the death certificate.[35] (The graph shows the death rate per 100,000 population for white males between 50 and 69 years of age for four levels of particulate matter, Buffalo and environs, 1959-1961.)

Among white males and females between 50 and 69 years of age, the death rate from gastric carcinoma was 27 per 100,000 population for those living in the area with the least suspended particulates, half again as high for those living in the next most polluted area, and one and one-half times as high for those living in the most polluted area. (See Table 11-4 and Figure 11-6.[35])

The Nashville Air Pollution Study by Hagstrom et al.[38] and Zeidberg et al.[39] used sulfation and dustfall data from 123 sampling stations, and sulfur dioxide concentrations and soiling indices from 36 stations. All codable deaths registered between 1949 and 1969 (32,067) were then

Table 11-4. AVERAGE ANNUAL DEATH RATES PER 100,000 POPULATION FOR GASTRIC CANCER ACCORDING TO ECONOMIC AND PARTICULATE LEVELS; WHITE MALES, 50-69 YEARS OF AGE, BUFFALO AND ENVIRONS, 1959-1961.[35]

Economic Level	Particulate Level				
	1 (Low)	2	3	4 (High)	Total
1 (low)	—	0(0)	63(10)	136(8)	77(18)
2	45(5)	41(12)	48(10)	84(8)	50(35)
3	—	39(9)	51(6)	51(2)	44(17)
4	15(3)	38(9)	63(5)	—	33(17)
5 (high)	26(5)	16(3)	0(0)	—	20(8)
Total	26(13)	34(33)	53(31)	—	42(95)

Figures in parentheses indicate numbers of deaths

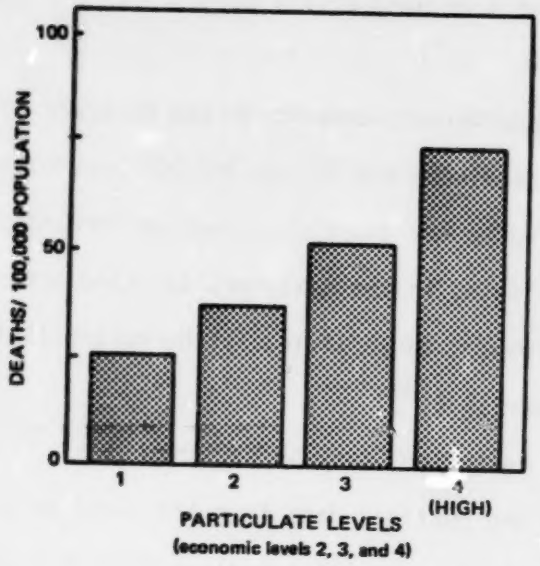

Figure 11-6. Average annual death rate from gastric cancer.[35] (The graph shows the death rate per 100,000 population for white males between 50 and 69 years of age for four levels of average particle concentrations, Buffalo and environs, 1959-1961.)

distributed among census tracts rated according to high, moderate, and low pollution levels, and upper, middle, and lower economic classes, and then further coded by age, sex, race, and underlying cause of death. Standardized mortality ratios (for total respiratory disease and for pneumonia, influenza, bronchitis, emphysema, tuberculosis, and lung and bronchial cancer) were then related to the pollution indices obtained during 1959. The statistically significant mortality increases were those for all respirtory diseases related to sulfation and soiling; lung and bronchial cancer mortality, and bronchitis and emphysema mortality were not clearly related to pollution indices. "High" pollution in these studies referred to soiling at more than 1.1 coh units/1,000 linear feet and SO_2 concentrations of more than about 30 $\mu g/m^3$(0.1 ppm). A later paper[40] derived from the same study period, analyzed infant and fetal death rates between 1955 and 1960. For white infant mortality, significant regressions were obtained for sulfation; dustfall (alone or as an interaction variable) was the most frequently related variable. No data were collected on smoking habits for this Nashville study. It should be noted also that the "middle class" group covered a relatively large segment of the decendents.

It is interesting to note that the association between suspended particle concentrations and gastric cancer in the Buffalo study, which appeared to be independent of economic status, was observed also in the Nashville study.[38]

c. Geographic Variations in Morbidity--Special Studies

It has been postulated that the study of records of illness rather than mortality should be more fruitful in defining subtle effects, since morbidity is an earlier and more sensitive index of deviation from normal health. A much larger insult presumably must be given to the body to cause death than to cause illness. However, routinely collected morbidity data are not generally available. Data occasionally may be obtained from group insurance statistics, hospital admission records, or existing school records. Since such data usually are not collected in a uniform, precise manner, most morbidity studies require expensive and time-consuming field surveys with questionnaires or actual medical examinations of the subjects.

Morbidity studies of adults involving long-term exposures are frequently not as useful as desired due to the presence of complicating factors such as occupation and smoking. Accordingly, Anderson[32] has recommended that children and housewives be used to determine the health effects of air pollution.

A study was conducted by Petrilli et al.[41] in Genoa, Italy, which followed Anderson's recommendation. The subjects were women over 65 years of age, nonsmokers who had lived for a long period in the same area and who had no industrial work experience. Economic and social conditions in the areas of residence were thus considered as were the levels of pollution in the areas of their residence. Pollutant measurements were carried out between 1954 and 1964 in 19 different areas, and morbidity indices were calculated for 1961 and 1962 for this population, which received free medical care from the municipality and therefore wasunder continuous medical observation. There was a significant correlation between the frequency of bronchitis with mean annual sulfur dioxide levels (r=0.98), and a nonsignificant correlation for mean annual suspended particle concentrations (r=0.82) and mean annual dustfall (r=0.66).

Toyama,[42] in a comprehensive study of air pollution and its health effects in Japan, charts the age-standardized morbidity rates (per 1,000) secured by interview survey in 1961 and describes a gradient of respiratory disease morbidity from the highly industrialized (and presumably polluted) areas to the rural areas of Japan. Further, the pulmonary disease morbidity rate was higher in the industrialized, polluted areas than were the rates for other disease groupings. The gradient was not noted for cardiovascular diseases nor for gastrointestinal diseases. Unfortunately, clear statements of specific pollutant concentrations do not accompany these data on morbidity. The age-standardized morbidity rates per 1,000 for several cities in Japan are shown in Figure 11-7.

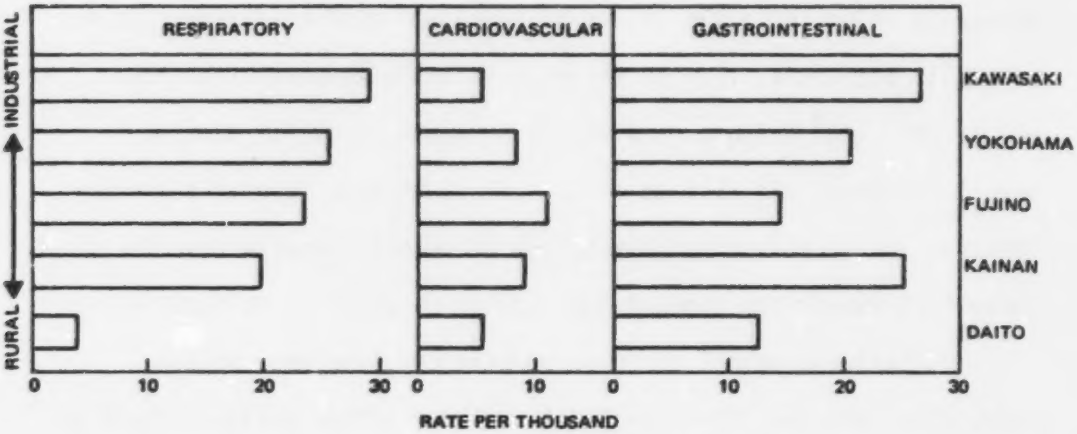

Figure 11-7. Age-standardized morbidity rate per 1,000 for three types of diseases in Japan.[42] (The mortality rate for respiratory diseases shows a clear correlation with pollution levels; there is no such clear correlation for cardiovascular or gastrointestinal disorders.)

Holland et al.[43] studied the prevalence of chronic respiratory disease symptoms and performance of pulmonary function tests in a comparative study of outdoor telephone workmen in London, in rural England, and on the east and west coasts of the United States. Types of occupational exposure, use of cigarettes, and socioeconomic matching were considered. The annual mean concentrations of suspended particulate matter in the British exposures were approximately 200 $\mu g/m^3$, and approximately 120 $\mu g/m^3$ in the American case. For sulfur dioxide, the mean concentrations were: in London 300 $\mu g/m^3$ (0.1 ppm), in rural areas of England 60 $\mu g/m^3$ (0.02 ppm), and in the United States between 30 and 120 $\mu g/m^3$ (0.01 and 0.04 ppm). Persistent cough, phelgm, and chest illness episodes and increased sputum volume were all significantly more frequent in men (between the ages of 50 and 59) in the London area than in rural England; and pulmonary function was poorer in relation to the levels of smoke and sulfur dioxide. It is possible, therefore, than an increase in smoke concentration from 120 to 200 $\mu g/m^3$ (with an equivalent increase in sulfur dioxide level) increases the risk to older workers of deteriorated pulmonary function and increased symptomatology of chronic respiratory disease. Table 11-5 summarizes some of the results obtained by Holland.

Holland and Reid[44] reviewed respiratory symptoms, sputum production, and lung function levels in post-office employees both in central London and in peripheral towns. Over the age of 50, the London men had more frequent andmore severe respiratory symptoms, produced more sputum, and had significantly lower lung function tests. Socio-economic factors were presumed the same, the occupational exposures were homogeneous, and corrections were applied for smoking. There were

Table 11-5. RESPONSE OF TELEPHONE WORKERS IN THE UNITED KINGDOM AND UNITED STATES TO AIR POLLUTION[43]

	United Kingdom				United States			
	London		Rural areas		Baltimore Washington Westchester County		San Francisco Los Angeles	
	Age 40–49	Age 50–59	Age 40–49	Age 50–59	Age 40–49	Age 50–59	Age 40–49	Age 50–59
Number of men examined	113	137	267	159	396	229	361	119
Persistent cough and phlegm (percent)	25.7	38.7	24.0	18.9	22.2	25.8	21.6	24.4
Persistent cough and phlegm and chest illness episode (percent)	10.6	10.9	7.5	5.0	6.8	7.0	4.0	7.6
$FEV_{1.0}$ (liters) mean values:								
Non smokers	2.8	2.6	3.0	2.8	3.5	3.1	3.7	3.3
Cigarette smokers:								
1–14 per day	2.6	2.3	2.8	2.6	3.4	3.0	-------	-------
15–24 per day	2.6	2.2	2.8	2.5	3.2	2.8	3.4	2.8
25 or more per day	2.5	2.1	2.7	2.5	3.2	2.9	-------	-------
Sputum volume 2 cc. or more (percent)	28.9	42.9	22.1	23.5	7.1	10.0	8.6	14.3
Suspended particulate matter, 24 hr ($\mu g/m^3$):								
Mean	220		200		120		120	
Maximum	4000		3000		500		340	
Sulfur dioxide, 24 hr ($\mu g/m^3$), ppm in parentheses:								
Mean	290 (0.1)		60 (0.02)		110 (0.04)		30 (0.01)	
Maximum	3700 (1.3)		740 (0.26)		715 (0.25)		170 (0.06)	

some physique differences among people in the rural areas; allowances were made for these in the statistical evaluation. Unfortunately, no quantitative air quality determinations accompanied these results. The authors nevertheless concluded that the most likely cause of the difference in respiratory morbidity between the men working in central London and those in the three rural areas was related to the differences in the local air pollution. On the other hand, studies on Jamaicans dying from emphysema revealed no differences between rural and urban dwellers; however, the pollution levels were not given. The small differences observed between East Indians and the other racial groups examined were related to cigarette smoking rather than generalized atmospheric pollution.[45]

In a group of Canadian veterans studied by Bates[46] a relationship between air pollution and both bronchitis and pulmonary function measurements has been reported. Unfortunately, the data on the levels of smoke, sulfur dioxide, and other pollutants in the four Canadian cities compared are inadequate to derive specific relationships between these pollutants and the prevalence or exacerbation of disease of the deterioration of pulmonary function. However, there is an association in the "dirty" cities (Montreal and Toronto) versus the "clean" cities (Halifax and Winnipeg) of increased prevalence and severity of bronchitis and of poorer pulmonary functions.

Ferris and Anderson[47] and Anderson, Ferris, and Zickmantel[48] completed a comparison of respiratory disease incidence and air pollution in three residential areas of Berlin, New Hampshire, by prevalence survey. Subjects who were nonsmokers and those who were moderate smokers were separately evaluated. Monthly dustfall and sulfur dioxide concentrations were recorded at one or more stations in each area. Prevalence of respiratory disease and pulmonary function ($FEV_{1.0}$ and peak expiratory flow rate) determinations were made. The mobility of the population, variations of concentrations of pollutants within a given area, possible effects from the area of occupation, and differences in the in-home environment were considered as contributors to the lack of consistent relationships between the health effects and the measured parameters of air pollution. In a later study Anderson and Ferris[49] compared this "polluted" town in New Hampshire with a control community in British Columbia and again found an obscuring of the effects of air pollution on respiratory symptoms and pulmonary function suggesting perhaps the overwhelming effect of cigarette smoking in symptom production. It should be noted, however, that the pollution levels and the ranges were possibly too low or too small to show a relationship.

The Nashville air pollution study[50] reviewed total morbidity in relation to air pollution. A wide matrix of sampling stations was used to group the residences of individuals into areas of high, moderate, and low pollution according to annual geometric means of measurements. The morbidity data were secured by home interview. Significant "direct correlations" of total morbidity with levels of pollution (measured by soiling index and 24-hour sulfur dioxide levels) were observed for individuals over 55 in the middle socioeconomic class. Cardiovascular diseases were directly correlated with the aerometric parameters. There was no statistical evidence of increased respiratory disease morbidity or morbidity of other organs and systems. The same qualifications noted for the mortality data of this study apply to these data on morbidity (see section C-2-b-(2)).

d. Morbidity--Incapacity for Work

Dohan[51] reviewed the incidence of respiratory disease producing absence from work in a population of women employed in several branches of an electronics company in the eastern part of the United States. Pay scales were roughly equivalent and presumptions were made, therefore, that the socioeconomic status and smoking patterns were uniform. Air measurements were made from areas near the town of employment (for suspended particulate sulfates, nitrates, copper, zinc, vanadium, nickel, and acetone-soluble organic matter, as well as total suspended particulate matter). Absences for a respiratory illness in excess of 7 days were calculable from company employment records and health insurance records. There was a significant correlation of respiratory disease absence frequency with the concentration of suspended particulate sulfates, based on 24-hour values measured biweekly; although the level of total suspended particulate matter ranged from 100 to 190 $\mu g/m^3$ in the various cities, a correlation was not demonstrated. The implication of the significantly correlated datum "suspended particulate sulfate" is not clear, as it might, for example, be an index of the community fuel consumption or an index of sulfur dioxide irritation.

Burn and Pemberton[34] reviewed the increased number of certificates of incapacity issued to workers in Salford in relation to smog episodes. When mean daily smoke pollution, based on data from five sampling stations, exceeded 1,000 $\mu g/m^3$ for 2 consecutive days, the number of "bronchitis certificates" issued exceeded the expected number by a factor of 2, on four of five occasions in 1958.

During 1961-1962 a study of the incidence of incapacity for work was conducted by the British Ministry of Pensions and National Insurance.[52] The population covered was representative of the working population of England and Wales. Rates of sickness absences for bronchitis, influenza, arthritis, and rheumatism were related to indices of pollution. There was a significant correlation between bronchitis incapacity in middle-aged men (35-54) and the average seasonal (October through March) concentrations of smoke and sulfur dioxide in high density residential districts, based on 24-hour measurements. For Greater London, there was a significant correlation between bronchitis incapacity and both smoke and sulfur dioxide for all age groups taken together, and for men aged 35 to 54 and 55 to 59. It is interesting that there was also more incapacity from arthritis and rheumatism in areas having heavy smoke pollution. Influenza incapacity was greater in those areas with higher pollution levels over Great Britain as a whole but not within the Greater London conurbation, nor was there in this latter area any association between pollution and psychosis or psychoneurosis. The lowest bronchitis inception rates related to smoke levels between 100 and 200 $\mu g/m^3$ and to sulfur dioxide concentrations between 150 and 250 $\mu g/m^3$ (0.053 and 0.081 ppm). The highest values related to particle concentrations of 400 $\mu g/m^3$ and sulfur dioxide concentrations of 400 $\mu g/m^3$ (0.14 ppm).

Lowe et al.[53] from a study of a group of workers have conluded that insofar as the etiology of chronic bronchitis is concerned among steel workers, sulfur dioxide and exposure to steel mill grit are not as important as cigarette smoking.

Verma et al.[54] presented information on illness absences in relation to air pollution. Illness data for the employees (males and females, aged 16 through 64) of a metropolitan New York insurance company were obtained through the records of the personnel department. They included medical history, X-ray information, and laboratory results obtained by the medical department of the company. Absences were classified as those due to respiratory illness and those due to non-respiratory illness. Mean daily concentrations of air pollution and meteorological data were secured from the monitoring system of metropolitan New York: smoke shade, sulfur dioxide, and carbon monoxide content were reported. The data for the 2 years, 1965 and 1966, were examined statistically and several conclusions were reached. There was a strong time dependence, and yearly cyclical behavior; when this factor was removed there remained no strong positive relationship between respiratory-caused absence and the pollution variables studied. Respiratory illness absence rates were highest when sulfur dioxide and smoke shade levels were both high on cool days; a lag effect for repiratory absences was not noted.

3. Studies of Children

Comparisons of the prevalence of respiratory disease in areas of different average pollution concentrations have been made to delineate the role of air pollution and specific pollutants. A problem common to all the studies is the difficulty in guaranteeing that the areas are similar (except for air pollution) in all factors that might affect the prevalence of disease.

Because studies on adults tend to be complicated by smoking habits, changes of occupations, and changes in address over a period of years, several studies have been directed at effects of air pollution on school children. The advantages of utilizing children for research on the primary etiologic effects of air pollutiowere first noted by Reid[55] several years ago; Anderson[32] has reaffirmed this view. A major element of concern is that deleterious effects on the respiratory system of very young children may have an effect on the subsequent evolution of the chronic bronchitis syndrome in the adult population.

The relationships of respiratory infections to long-term residence in specific localities have been studied in England by Douglas and Waller.[56] Indices of air pollution, in terms of domestic coal consumption records, were used to classify four groups; the authors included an evaluation of the validity of this method at the end of their report. The histories of 3,866 children born during the first week of March 1946 were followed until 1961, when the children were 15 years of age. Social class composition of these children did not differ significantly from area to area. Measured concentrations for smoke and for SO_2 in 1962 and 1963 were compared with the earlier prediction of pollution intensity based on the coal consumption data, and indicated an overlap for the Greater London area of low and moderate groupings; for other areas, the predicted gradient of concentrations was affirmed. Because of the age of the subjects, smoking was apparently not considered in this evaluation. In 1965, 19 percent of the boys and 5 percent of the girls, aged 11 to 13, smoked at least one cigarette a week regularly.[57]

In the Douglas and Waller study,[56] the generation of pollutants in the indoor home environment (e.g., by heating and cooking) was not considered. Interviews were conducted with the mothers when the children were 2 and 4 years of age; information was obtained about upper and lower respiratory illness and recorded hospital admissions for these and other causes. Data about colds, coughs, and hospital admissions were also gathered by school doctors at medical examinations when the children were 6, 7, 11, and 15 years of age. Between the ages of 6½ and 10½, special records for causes of school absences exceeding one week were reviewed with the mothers. A total of 3,131 families remained in the same pollution area throughout the first 11 years of this study. The conclusions of the study were that upper respiratory tract infections were not related to the amount of air pollution, but that lower respiratory tract infections were. Frequency and severity of lower respiratory tract infections increased with the amount of air pollution exposure, affecting both boys and girls, and with no differences detectable between children of middle class and working class families. This association was found at each of the examination ages, including age 15. At age 15, persistence of rales and rhonchi (chest noises), possibly the prodrome of adult chronic respiratory disease, was some tenfold less in the very low pollution area and a factor of two less in the low pollution area than that in the high pollution area. If the 1962-1963 measured concentrations for smoke and SO_2 can truly be extrapolated to the 15-year respiratory illness survey, then these British school children experienced increased frequency and severity of lower respiratory diseases in association with annual mean smoke concentrations ranging above 130 $\mu g/m^3$ and with SO_2 above 130 $\mu g/m^3$ (0.046 ppm).

The lower respiratory tract findings of Douglas and Waller were confirmed in a study by Lunn et al.[58] The patterns of respiratory illness in school children of the age group 5 to 6 have been studied with reference to residence in four areas of Sheffield. Mean daily particle measurements in each of four areas ranged from 97 $\mu g/m^3$ in the "low" area to 301 $\mu g/m^3$ in the "high" area; in the two extreme areas, SO_2 concentrations were respectively 123 and 275 $\mu g/m^3$ (0.043 and 0.096 ppm) during 1963-1964. Somewhat lower pollution levels were noted the following year, although the gradient between the districts was preserved.

Questionnaire to the parents, physical examination, observation for the presence of nasal discharge, examination of the eardrums, and recording of both the forced expiratory volume of 0.75 seconds ($FEV_{0.75}$) and the forced vital capacity (FVC) were completed during each of the summer terms of 1963, 1964, and 1965. Several socioeconomic factors were compared for the various districts; smoking was appropriately disregarded for this age group; internal home environments, or differences in home heating systems, were not reported. The authors conclude that there is an association with the levels of atmospheric pollution and chronic upper respiratory infections (as indicated by mucopurulent nasal discharge, history of three or more colds yearly, or scarred or perforated eardrum). Further, lower respiratory tract illness (measured by history of frequent chest colds or episodes of bronchitis or pneumonia) was similarly associated. Functional changes, in the form of reduced $FEV_{0.75}$ ratios emerged where there was a past history of pneumonia or bronchitis, of persistent or frequent cough, or of colds going to the chest. From these changes, there appears to be a persistence of respiratory dysfunction, even in the absence of high pollution at the time of the function testing. The lowest "effect" level for smoke and SO_2 is not clearly indicated by this study, but the increased association of "respiratory infections" in school children can be detected for areas where mean daily figures exceed about 100 $\mu g/m^3$ for smoke and 120 $\mu g/m^3$ (0.042 ppm) for SO_2.

The exacerbation of acute respiratory illness of school children in Ferrara, Italy, has been studied by Paccagnella et al.[59] Air pollution measurements from 1959 through 1964 permitted definition of high, medium, and low zones of pollution. School children in the age range 7 to 12 had daily examinations, and the date of onset of acute respiratory disease was recorded. Although weather conditions were related to changes in pollutant levels, the onset of acute respiratory disease in the children was not correlated significantly with changing air pollution values, except in the poorest socioeconomic area. The pollutant values for this community are, however, lower than those encountered in the British studies previously discussed (20 to 45 $\mu g/m^3$ annual mean particle concentrations).

Toyama[42] studied two groups of school children, 10 to 11 years old, in Kawasaki, Japan. Sulfation rates at the school in the more polluted area varied from 0.5 to 1.9 and averaged 0.9 $mg/100cm^2$-day PbO_2; no sulfation rates were given for the school in the area of lower pollution. Dustfall was considerably less in the area of lower pollution (ranging from about 5 to 15 $tons/km^2$-mo) than in the more polluted area (ranging from about 15 to 70 $tons/km^2$-mo). The children from the more polluted area had a higher frequency of nonproductive cough, irritation of the upper respiratory tract, and increased mucus secretion. Whether the effect was due to oxides of sulfur or particulate matter cannot be determined from this study.

Shy et al.[60] in studying children in Chattanooga, Tennessee, found that ventilatory performance of second grade school children in a high-NO_2 area was significantly lower than the performance of children in the control area. Suspended nitrate particles did not have this effect. Respiratory illness rates were consistently higher in all family segments in the high-NO_2 exposure area when averaged for the entire 24 weeks of study. Shy et al.[61] found a relative excess in respiratory illness of 18.8 percent among families exposed to elevated NO_2 concentrations and an excess of 10.4 percent among families in the elevated suspended particulate exposure area.

A study by Manzhenko[62] of upper respiratory tract conditions in school children in Irkutsk is difficult to relate to the Sheffield study. However, the higher incidence of respiratory tract conditions and the undefined abnormal X-ray findings in these children's lungs are disturbing evidence of the possibility of an association between serious respiratory disease and residence in a polluted community.

4. Studies of Pulmonary Function

Holland et al.[43] reported decreased performance of the $FEV_{1.0}$ test in London and British rural outside telephone workers compared with their American counterparts. For both groups, the FEV was further decreased in relation to smoking intensity. FEV differences within the United Kingdom workers (i.e., London versus rural) could also be related to the sulfur dioxide concentrations accompanying the particle concentrations. A more detailed discussion of the study appears in section C-2-c.

Toyama[42] reported measurements of peak flow rate and total vital capacity performance in Japanese school children in areas of differing air pollution, measured monthly; fluctuations were observed in the mean peak flow rates of children attending schools and living in polluted industrial areas, the variations were smaller for children in clearer areas. Total vital capacity was not significantly different between pupils of the various schools. There was a substantial difference in peak flow rates between the two school districts at times of highest pollution. When pollution values were lowest, the differences were less. The lowest values related to dustfall measurements of 60 tons/km^2-mo and daily sulfur dioxide levels equivalent to 20 µg/cm^2 (lead peroxide candle measurements).

In Osaka, Watanabe[20] studied the peak flow rate and vital capacity performances by the children of schools enduring differing air pollutant concentrations. It was noted that individual peak flow rates were more markedly decreased in the winter months (September to December 1963) for the school in the highly polluted area than for the school in the low pollution area. Daily mean concentrations of both dustfall and sulfur dioxide were twice as great in the polluted area as in the unpolluted area.

In a comparison by Prindle et al.[63] of pulmonary function and other parameters in two Pennsylvania communities with widely different air pollution levels, average airway resistance and specific airway resistance were measured in persons 30 years of age and older. These measurements were performed at several stations in each of the communities and indicated differences between the high pollution and low pollution communities which were probably related to the sixfold difference in dustfall amounts. However, smoking and occupation were not accounted for.

In the paper by Lunn et al.,[58] Sheffield school children were shown to have reduced $FEV_{0.75}$ and FVC ratios in the area of highest pollution. The measurements were made during the summer, when pollution levels were low and apparent incidence of acute respiratory infection was diminished, suggesting, in contrast to the Japanese studies referred to above, that there may be persistence of the respiratory function deterioration in relation to residence in the area of high pollution. Mean daily averages measured at a single station were smoke 300 $\mu g/m^3$ and SO_2 275 $\mu g/m^3$ (0.096 ppm). A followup study on these same children in 1970, plus an additional group of 1,049 eleven-year-old children, indicated that the former group had less respiratory illness than the latter. It was concluded that overall improvement in pollution levels in Sheffield had resulted in the decrease in respiratory illness.[64]

The studies relating morbidity and deterioration in pulmonary function to particulate levels cover effects which are also included in Level III of the World Health Organization's "guides to air quality."[23]

5. Studies of Panels of Bronchitis Patients

Lawther[65] and Waller and Lawther[66] related several episodes of acute urban pollution to worsening of condition in a group of well-studied bronchitic patients at St. Bartholomew's hospital. Changes in their symptoms were recorded in a daily diary and acute worsening in significant numbers of the group was associated with daily rises in air pollution above 3 μg/^{3}m of smoke and 600 μg/m^3 (0.21 ppm) of sulfur dioxide. Figure 11-8 shows graphically the effects observed on 29 bronchitic patients during periods of high pollution in January 1954.

Angel et al.[67] reviewed the occurrence of new respiratory symptoms in men, working in factories and in offices, most of whom had prior evidence of chronic bronchitis. The study group of 85 men observed through the winter of 1962-1963 was selected from a group of 1,000 men aged 30 through 59, without apparent classification of either smoking patterns or possible occupational or residential exposure differences. Increased sputum production, deterioration of pulmonary function performance ($FEV_{1.0}$) and the more frequent occurrence of respiratory symptoms classified as "upper" (coryza, influenza, and acute respiratory disease), and "lower" (chest colds, bronchitis, wheezy attacks, pneumonia) were all associated with increased in both smoke and sulfur dioxide concentrations. There was frequent difficulty in defining an exacerbation of disease in those individuals already experiencing chronic bronchitis. During this period, illness peaks (attack rates) may have occurred with weekly mean concentrations of smoke exceeding 400 μg/m^3 and of sulfur

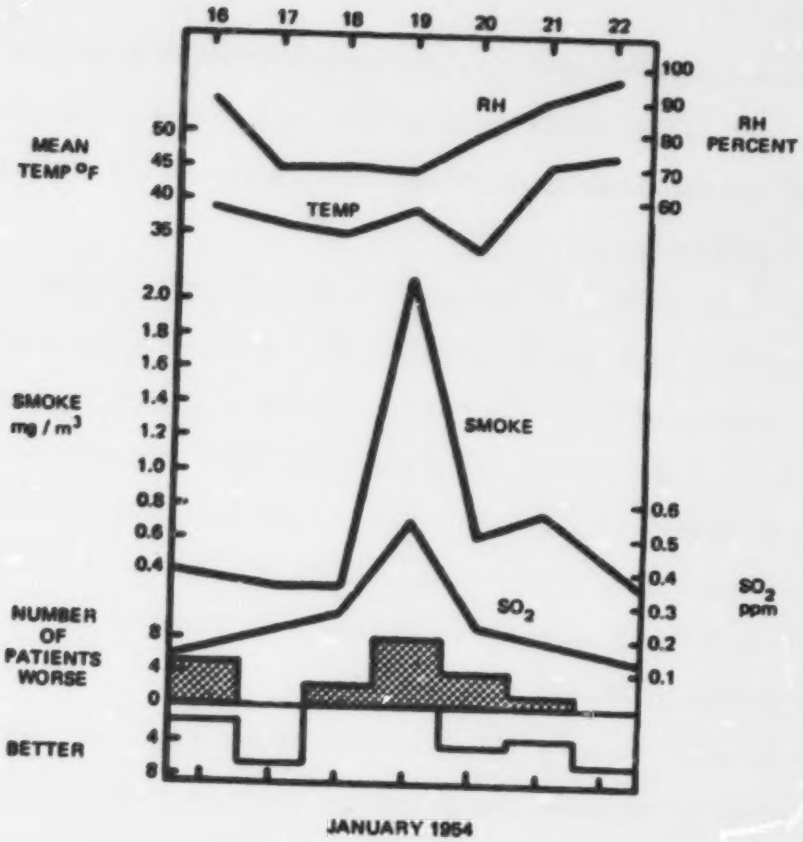

Figure 11-8. Effect on bronchitic patients of high pollution levels (January 1954).[65,66] (The figure represents the effect on bronchitic patients of increased pollution levels; patients stated whether they regarded their condition as "worse" or "better.")

dioxide exceeding 460 μg/m^3 (0.16 ppm); weekly mean concentrations were calculated using the highest daily mean occurring each week at each of 13 locations in the area.

Biersteker[68] surveyed male municipal employees in Rotterdam for symptoms of bronchitis and for peak flowmeter performance, and reviewed years of residence in Rotterdam versus years of residence in a nonurban environment as well as smoking habits. The individuals with bronchitis symptoms were matched with individuals without such symptoms but with similar age and background. Significant differences related to heavy cigarette smoking, but no reliable statistical indication of an effect of

First hour sputum volume--In men of constant smoking habits[69]

	1961	1962	1963	1964	1965	1966
Summer..........	1.65	0.83	0.93	0.93	0.83	0.72
Winter..........	1.38	1.27	1.20	1.13	0.70	....

urban or nonurban residence in the production of symptoms was detectable. Since particle concentrations in Rotterday are very low, the difference between concentrations in Rotterdam and in the nonurban environment may have been almost undetectable.

Fletcher et al.[69] followed 1,136 working men aged 30 to 59 in west London by surveys at 6-month intervals. The surveys included collection and measurement of morning sputum volume, FEV, and respiratory symptom questionnaires. While expected patterns of decline in lung function with age occurred (and this was the most rapid in cigarette smokers with low lung function to start with), an unexpected finding was a decrease in sputum volume in men with constant smoking habits. This was most consistent in the winter samples, but showed a significant trend in samples taken together as well as in the winter samples. (Trend data are based on 825 men who attended at least nine periodic examinations.) The authors felt this was possibly due to a decline in air pollution in London. They presented data on trends in SO_2 and black suspended matter which indicated that there is a consistent downward trend in particulate pollution (highly significant) but a less steady decline in SO_2.

This unique set of data could mean that with a decrease of smoke pollution (yearly mean) from 140 to 60 $\mu g/m^3$, there is an associated decrease in mean sputum volume during the first morning hour from about 1.5 to 0.75 ml. A contribution to this effect by the decrease in SO_2 is less likely than the entire effect resulting from the decrease in smoke. Possible changes in cigarette tars, in methods of smoking, and in techniques for collecting a sample, or other factors, could have influenced this result.

D. SUMMARY

This chapter reviews epidemiological studies of the relationship between pollutant concentrations and their effects on health. Indices varying from disturbance of lung function to death are considered. Concentration measurements of pollutants are sometimes available for the period during which the indicators were under review, and in these cases it is possible to state pollutant levels at which an effect is noted. However, such values cannot be taken to mean that effects will not be noted at lower concentrations. Most studies involving long-term effects using a geographic area comparison will use the cleanest area as the "control" area against which mortality and morbidity differentials are to be found. This necessarily assumes that this "cleanest" area itself does not have any effects from its level of pollution. Conversely, some studies have compared areas with very low particulate loading or with very limited ranges of differences. It is not possible to demonstrate any relationship under conditions of minimum values or minimal differences. Table 11-6 summarizes the epidemiological studies reviewed according to the several indices.

From the material reviewed in this chapter, a selection of data has been made from those studies which furnish the best quantitative information. Pollution measurements are given in the system used in the

Table 11-6. SUMMARY TABLE OF EPIDEMIOLOGICAL STUDIES

Health Index	Suspended particulate matter µg/m³	Smoke or black suspended matter (B. S. M.) µg/m³	COH (in brackets) or dustfall tons/mi²-mo	SO_2 µg/m³ daily average bracketed values are sulfation rates in mg/100 cm²-day	Findings	Reference
A. Acute episodes—Mortality:						
1. London, winter 1958–59.	------	rise of 200	------	rise of 75	Correlation of daily mortality (all causes) and daily bronchitis mortality with B.S.M. Significant positive association of SO_2 and deaths.	11
London, winter 1958–59.	------	1000	------	715	Increase in mortality (all causes)	12
London, winter 1958–59.	------	750	------	715	Re-analysis of data of reference 4	13
2. London, fog episodes, 1954, 1955, 1956, 1957.	------	2000	------	1200	Bronchitis death rate increased, other causes rise also.	8,9
London, fog episode 1962.	------	2000	------	1145	Death rates of elderly rose; greater proportionate rise in bronchitis deaths.	10
3. New York, November, 1953.	------	-----------	[5]	2460	Excess deaths all age groups; lag effect noted.	14
4. New York, December, 1962.	------	-----------	------	-----------	No excess deaths...............	15
New York, December, 1962.	------	-----------	[6]	2145	Excess deaths in 45–64 age group and over 65 age group.	16
5. New York, January, 1963.	------	-----------	[6]	1715	Peak in death rate superimposed on high deaths due to influenza.	16
6. Detroit, September, 1952.	>200 3 days	-----------	------	2860 (max)	Excess infant mortality and cancer mortality.	19
7. Osaka, December, 1962.	1000 (sic)	-----------	------	285	Sixty excess deaths...............	20
B. Acute episodes—Morbidity:						
1. New York, November, 1953.	------	-----------	[3]	715	Increased upper respiratory infection and cardiac disease morbidity.	21
2. New York, November, 1966.	------	-----------	------	-----------	Increased emergency visits for bronchitis and asthma; analysis complicated by Thanksgiving holiday.	22
C. Long-term air pollution—Day to day variations in mortality and morbidity:						
1. London winters, 1958–59, 1959–60.	------	>500	------	>400	Increases in mortality and morbidity appear positively associated with fluctuations in air pollution.	12

Table 11-6 (continued). SUMMARY TABLE OF EPIDEMIOLOGICAL STUDIES

Health Index	Suspended particulate matter µg/m³	Smoke or black suspended matter (B. S. M.) µg/m³	COH (in brackets) or dustfall tons/mi²-mo	SO_2 µg/m³ daily average bracketed values are sulfation rates in mg/100 cm²-day	Findings	Reference
2. New York City 1962–64.	------	-----------	------	-----------	Some periods of high air pollution appear to be related to air pollution; relationship not clear during other periods of high air pollution.	16
3. New York City, housing project.	------	-----------	------	-----------	Cough symptoms related to rise in air pollution in general; eye irritation related to rise in SO_2.	22
D. Long-term air pollution—Geographic variations in mortality:						
1. U.K., 214 areas, 1955–59.	------	200	------	200	Excess bronchitis mortality. Social index also important. Pollutant concentrations not measured in same period as mortality.	26
2. U.K., 1950–54 lung cancer; 1950–53 bronchitis, pneumonia; 1954–57 sampling network data.	n.a.	-----------	------	n.a.	Lung cancer mortality, bronchitis, and pneumonia mortality in males and bronchitis in females, strongly correlated with smoke density. Cancer of stomach and intestine correlates for some areas.	27–31
U.K., 1950–54 lung cancer; 1950–53 bronchitis, pneumonia; 1954–57 sampling network data.	------	-----------	------	-----------	Smoke, occupational, and population parameters relate to lung cancer mortality. Vanadium contributes to lung, female cancer, pneumonia mortalities. Pollutant measurements do not parallel time period of mortality studies.	
3. Eston, England.	------	160	------	115	Positive association between the degree of air pollution and the incidence of both bronchitis and lung cancer.	33
4. Salford, U.K., 3 wards, August, 1957, through.	------	"High": winter 680, summer 270 "Low": winter 450, summer 170.	------	"High" winter 750, summer 270 "Low": winter 360, summer 150.	High rate of bronchitis and lung cancer mortality. Death from all causes increased in "high" compared to "low" pollution wards.	34
5. Buffalo, N.Y., areas July, 1961–June, 1963.	100	-----------	------	[SO_3 0.45]	Positive correlation of pollutant levels with chronic respiratory disease deaths, and deaths from all causes. Mortality rates for gastric cancer 2 times higher in high particulate areas as compared to low particulate area.	35–37

Talbe 11-6 (continued). SUMMARY TABLE OF EPIDEMIOLOGICAL STUDIES

Health Index	Suspended particulate matter µg/m³	Smoke or black suspended matter (B. S. M.) µg/m³	COH (in brackets) or dustfall tons/mi²-mo	SO_2 µg/m³ daily average bracketed values are sulfation rates in mg/100 cm²-day	Findings	Reference
					Air pollution levels did not relate to deaths due to cancer of bronchus, trachea or lung. Mortality effects also seen in area with 80 µg/m³ to 100 µg/m³.	
6. Nashville, Tenn., 1940–60.	------	-----------	[>1.1]	80	"High" pollution related to increased respiratory disease mortality. Lung and bronchial cancer, bronchitis, and emphysema mortality not clearly related.	38,39
E. Long-term air pollution—Geographic variations in morbidity:						
1. Genoa, Italy	------	-----------	------	-----------	Frequency of bronchitis significantly associated with SO_2, not with suspended matter of dustfall.	41
2. Japan	------	n.a.	------	n.a.	Gradient of respiratory disease morbidity from industrial to rural sites.	42
3. U.K.-U.S. Comparison.	U.S. 100	U.K. 200	------	U.S. 12–30 U.K. 60–300	Increase of smoke concentration from 100 µg/m³ to 200 µg/m³ and equivalent SO_2 increase leads to higher risk to older men of poorer lung function and increased chronic respiratory disease.	43
	(Levels not directly comparable)					
4. U.K.	------	-----------	[11.6] (1 yr. av.)	[SO_3 1.3]	Differences in area respiratory morbidities attributed by authors to difference in air pollution.	44
5. Canada, 4 cities.	Inadeq.	-----------	------	Inadeq.	Increased severe bronchitis, poor lung function performance in "dirty" cities versus "clean" cities.	47
6. Berlin, N. Hampshire, Chilliwack, B.C.	------	-----------	34.9	[SO_3 0.4] [SO_3 0.05]	No observable differences	48-50
7. Nashville, Tenn.	------	-----------	------	-----------	Cardiovascular disease directly correlated with aerometric data. No increased respiratory disease morbidity.	51
F. Long-term air pollution—Incapacity for work:						
1. U.S.	100–190	-----------	------	-----------	No correlation of absence in excess of 7 days with suspended particles. Significant correlation of absence with "suspended particulate sulfate."	52

Table 11-6 (continued). SUMMARY TABLE OF EPIDEMIOLOGICAL STUDIES

	Health Index	Suspended particulate matter μg/m³	Smoke or black suspended matter (B. S. M.) μg/m³	COH (in brackets) or dustfall tons/mi²-mo	SO_2 μg/m³ daily average bracketed values are sulfation rates in mg/100 cm²-day	Findings	Reference
	2. Salford, U.K.	------	>1000 (2 consecutive days)	------	-----------	Bronchitis caused absence greater than expected by factor of 2 in four of five episodes.	34
	3. Greater London, U.K.	------	100–200	------	150–250	Lowest bronchitis inception rates	54
			400		400	Highest bronchitis inception rates. Significant correlation of bronchitis incapacity with smoke and SO_2 for all age groups, and men 35–54 and 55–59.	
	4. New York, N.Y.	------	-----------	[2.7] (7 yr. mean).	540 (7 yr. mean).	No strong relation between respiratory illness absence and COH or SO_2.	56
	Studies of children:						
G.	1. U.K., 1946–61.	>130	-----------	------	>130	Increased frequency and severity of lower respiratory diseases in school children.	58
	2. Sheffield, U.K., 1963–65.	100	-----------	------	>120	Increased association of "respiratory infections" for school children age 5–6.	62
	3. Ferrara district, Italy, 1959–64.	n.a.	-----------	------	n.a.	No correlation of onset of acute respiratory illness to changing pollutant levels except in poorest socioeconomic area.	63
	4. Japan	------	-----------	60	[SO_3 0.02]	Total vital capacity of children in polluted and nonpolluted areas the same. Fluctuations in mean peak flow rates larger for children in polluted areas than in less polluted areas.	42
	5. Japan	------	-----------	------	-----------	Peak flow rates decreased more in winter for children in polluted areas than in less polluted areas.	20
	6. Irkutsk, U.S.S.R., time period not definite.	n.a.	-----------	------	n.a.	Possible relation of respiratory disease and residence in area of pollution.	67
H.	Studies of pulmonary function:						
	1. U.K.-U.S. Comparison.	------	-----------	------	-----------	$FEV_{1.0}$ performance of U.K. workers decreased compared to U.S. counterparts.	43
	2. Japan	------	-----------	------	-----------	Total vital capacity of children in polluted and nonpolluted areas the same. Fluctuations in mean peak flow rates larger for children in polluted areas than in less polluted areas.	42

Table 11-6 (continued). SUMMARY TABLE OF EPIDEMIOLOGICAL STUDIES

Health Index	Suspended particulate matter $\mu g/m^3$	Smoke or black suspended matter (B. S. M.) $\mu g/m^3$	COH (in brackets) or dustfall tons/mi^2-mo	SO$_2$ $\mu g/m^3$ daily average bracketed values are sulfation rates in mg/100 cm^2-day	Findings	Reference
3. Japan	------	-----------	------	-----------	Peak flow rates decreased more in winter for children in polluted areas than in less polluted areas.	20
4. Pennsylvania, 2 communities.	------	-----------	------	-----------	Possible relation of dustfall and SO$_2$ differences in average and specific airway resistance of subjects in the two communities.	68
5. Sheffield, U.K.	------	100	------	275	Reduced FEV$_{0.75}$ and FVC in areas of highest pollution. Possible persistence of respiratory function deterioration related to residence in area of high pollution.	62
I. Studies of panels of bronchitic patients:						
1. London, November, 1955–May, 1956.	300	-----------	------	>600	Acute worsening of symptoms in bronchitis patients.	65
2. U.K., October, 1962–April, 1963.	400	-----------	------	460	Possible increase in respiratory disease attack rates.	67
3. Rotterdam, Netherlands.	------	n.a.	------	n.a.	No indication of residence effect on bronchitis symptoms.	68
4. London	------	140 $\mu g/m^3$ declining to 60 $\mu g/m^3$.	------	200 $\mu g/m^3$ declining to 160 $\mu g/m^3$.	Decrease in morning sputum volume with decreasing air pollution levels in bronchitis patients under observation during 6 years.	69

original observations, since conversion from one method to another is not recommended. Attention should be given to the difference between British "smoke" and American "suspended particulate" measurements. Both are given in micrograms per cubic meter of air, but they are not identical. Limited comparisons indicate that the American values may be higher in the same situation. In making use of these data, attention must also be given to the averaging time which was employed in the original observation. Long-term averages are, of necessity, considerably lower than selected high daily means in the same locations.

British studies of acute episodes of increased pollution show excess deaths occurring at smoke levels from 750 to 2,000 $\mu g/m^3$. High SO_2 levels are, of course, concurrently present. The excesses of mortality are always accompanied by a very large increase in illness, mainly exacerbations of chronic conditions. Similar but less spectacular episodes in New York City have been associated with smoke shade levels of 5 to 6 coh units.

Lawther,[65] in reviewing a long series of observations on the condition of bronchitic patients, estimated that they tended to become worse when daily levels of smoke exceeded 300 $\mu g/m^3$ with SO_2 over 600 $\mu g/m^3$. Angel et al.[67] made somewhat similar observations with smoke above 400 $\mu g/m^3$ and SO_2 over 460 $\mu g/m^3$.

Winkelstein[35-37] found in Buffalo that increases in the mortality rate were significantly linked to higher levels of suspended particulate pollution. His studies showed that mortality from all causes, from chronic respiratory diseases, and from gastric carcinoma increased from the lowest of his five levels of pollution (less than 80 $\mu g/m^3$) through the three higher ranges, after the effects of socioeconomic status had been considered. Zeidberg and coworkers [39,50] found in Nashville significant increases in all respiratory deaths at soiling levels over the 1.1-coh annual average. Neither of these studies took smoking habits into account, and the Nashville study only partially allowed for socioeconomic factors.

Studies of illness in relation to residence in more and less polluted areas contribute additional information. Fletcher et al.[69] noted a proportional decline in the production of morning sputum in chronic bronchitics in west London from 1961 to 1966 as smoke pollution in their residence areas declined from 140 $\mu g/m^3$ annual mean. Douglas and Waller[56] found an increase in frequency and severity of lower respiratory illness at smoke and SO_2 levels over 130 $\mu g/m^3$ annual average. The Sheffield study by Lunn et al.[58] shows similar differences occurring with some morbidity measures between about 100 and 200 $\mu g/m^3$ of smoke and for others between 200 and 300 $\mu g/m^3$, annual averages.

A study of British workmen found increased absences due to respiratory illness in areas with smoke levels in excess of 200 $\mu g/m^3$.

Physiologic studies of lung function have also been made in both adults and children. On the basis of present limited knowledge, it appears that the alterations found may be both temporary and permanent. The observations now available relate to long-term residence in a given area. The Sheffield study shows reduced pulmonary function in the children in the most polluted area--that is, where smoke concentration is above 300 $\mu g/m^3$. Studies in Japan show a decrease in pulmonary function in school children living in areas of high dustfall as compared with those living in low dustfall areas. In Osaka the dustfall levels were 6.5 and 13.3 gm/m^2-month.

The analyses of the numerous epidemiological studies discussed clearly indicate an association between air pollution, as measured by particulate matter accompanied by sulfur dioxide, and health effects of varying severity. This association is most firm for the short-term air pollution episodes.

There are probably no communities which do not contain a reservoir of individuals with impaired health who are prime targets for the effects of elevated levels of particulate matter and sulfur oxides. However, to show small changes in deaths associated with coincident higher levels of air pollutants requires extremely large populations. In small cities, these small changes cannot be detected statistically.

The epidemiological studies concerned with increased mortality also show increased morbidity. Again, increases in morbidity as measured, for example, by increases in hospital admissions or emergency clinic visits, are most easily detected in major urban areas.

It is believed that, for the large urban communities which are routinely exposed to relatively high levels of pollution, sound statistical analysis can detect with confidence the small changes in daily mortality which are associated with fluctuations in pollution concentrations. Unfortunately, thus far only limited analysis has been made.

The association between longer-term community exposures to particulate matter and respiratory disease incidence and prevalence rates is conservatively believed to be intermediate in its reliability. Because of the reenforcing nature of the studies conducted to date, the conclusions to be drawn from this type of study can be characterized as probable.

The association between long-term residence in a polluted area and chronic disease morbidity and mortality is somewhat more conjectural. However, in the absence of other explanations, the findings of increased morbidity and of increased death rates for selected causes, independent of economic status, must still be considered consequential.

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APPENDIXES

APPENDIX A—SYMBOLS

A	area of the filter stain, cm^2
B	turbidity coefficient (empirical), $\lambda = 0.5\mu$
C	particle concentration in the air, μg/m^3
B_o	turbidity coefficient, measured on the ground
B_z	turbidity coefficient, measured at height z above the ground
I	light intensity, usually in ergs/cm^2/sec
I_o	initial or incident light intensity
L_v	equivalent visual range, or visibility, but calculated on basis of a constant scattering coefficient, b, along the line of sight
P	probability
P(λ)	solar transmission factor for atmosphere aerosols; it is a function of λ
Q	particle mass concentration in the air, usually in μg/m^3
S	scattering cross-section, cm^2/particle
S_c	derived surface particle concentration, μg/cm^2
T	transmissivity, I/I$_o$
V	volume of air sampled, m^3
b	extinction coefficient, m^{-1} ($= b_{\text{abs aerosol}} + b_{\text{abs gas}} + b_{\text{Rayleigh}} + b_{\text{scat}}$)
$b_{\text{abs aerosol}}$	extinction coefficient due to absorption by aerosol particles
$b_{\text{abs gas}}$	extinction coefficient due to absorption by gas such as NO$_2$
b_{Rayleigh}	extinction coefficient due to scatter by gas molecules
b_{scat}	extinction coefficient due to scatter by aerosols
d	diameter of a spherical particle, cm
e	Napierian log base ($=2.7182818$)
g	acceleration of gravity, cm/sec^2
m	index of refraction
γ	correlation coefficient
v	settling velocity of a particle, cm/sec
x	distance, m or cm
z	height above ground, meters
α	light scattering coefficient (empirical) relates b to λ
β	turbidity coefficient for $\lambda = 1\mu$
ν	viscosity of air, or other gas, poises
λ	wavelength of light Angstroms, microns, or nanometers
χ	probability function, usually expressed as χ^2 ("Chi square")

APPENDIX B—ABBREVIATIONS

Å	Angstrom, $1\text{Å} = 10^{-8}$ cm
BTPS	body temperature, pressure, saturated
cm	centimeter, $1\text{cm} = 10^{-2}\text{m}$
cm^2	square centimeter
cm^3	cubic centimeter
CMD	count median diameter
coh	coefficient of haze
DMBA	10-dimethyl-1, 2 benzanthracene
FEV	forced expiratory volume
FVC	forced vital capacity
g	mass, grams
hr	hour
l	liter
m	length, meter
m^3	cubic meter
mg	milligram
mi	mile
mi^2	square mile
min	minute
ml	milliliter
MMD	mass median diameter
mo	month
mu	millimicron, $1\text{m}\mu = 0.001\mu$
μ	micron, $1\mu = 10^{-4}\text{cm} = 10^{-6}\text{m} = 10^{4}\text{Å}$
μg	microgram
ppm	parts per million
RUDS	reflectance unit of dirt shade
sec	second
yr	year

APPENDIX C—CONVERSION FACTORS

To convert—	*To—*	*Multiply by—*
mg/m³	µg/m³	1000
mg/100 m³	µg/m³	10
µg/ft³	µg/m³	35.314
µg/1000 m³	µg/m³	10^{-4}
µg SO_2/m³ (0°C, 760 mm Hg)	ppm SO_2 (vol)	3.5×10^{-4}
ppm SO_2 (vol)	SO_2 µg/m³ (0° C, 760 mm Hg)	2860
tons/mi²-mo	mg/cm²-mo	3.5×10^{-2}
tons/mi²-mo	g/m²-day	1.07×10^{-2}
tons/mi²-mo	metric tons/km²-mo	3.5×10^{-1}
tons/mi²-mo	µg/m²-mo	3.5×10^{5}
mg/cm²-mo	tons/mi²-mo	28.5
g/m²-day	tons/mi²-mo	85.5
µm	m	10^{-6}
µm	Å	10^{4}
Å	cm	10^{-8}
µm	cm	10^{-4}
bbl	gal.	55
1	ft³	0.0353
ft³	1	28.32
ml	in³	6.1×10^{-2}
m	ft	3.28
lb	g	453.6

Acid, free—an acid which is unneutralized by other compounds

Acid, stearic—the most common fatty acid occurring in natural animal and vegetable fats, almost completely colorless and odorless

Acrolein—a toxic colorless mobile liquid aldehyde with an acrid odor

Adenocarcinoma—a malignant tumor in which the cells are arranged in the form of glands or gland-like structures

Adenoma—an epithelial tumor, usually benign, with a gland-like structure

Adhesion—the attraction of two unlike substances

Adsorption—the phenomenon by which gases are attracted, concentrated, and retained at a boundary surface

Aerodynamics—a phase of the mechanics of fluids, its study being limited to the reactions caused by relative motion between fluid and solid, the fluid being limited to air in most cases but occasionally broadened to include any gas

Aerosol—a cloud of solid particles and/or liquid droplets smaller than 100μ in diameter, suspended in a gas

Aerosol, bidisperse—an aerosol in which all the suspended particles tend to be of two sizes (diameter)

Aerosol, monodisperse—an aerosol in which all the suspended particles are of nearly equal size (diameter)

Air, residual—the air that stays in the lungs after forceful expiration

Air, tidal—the air that is carried to and from the lungs during a respiratory cycle

Airway—any part of the respiratory tract through which air passes during breathing

Airway resistance—resistance to the flow of air in the passages to the lungs

Albedo—the ratio of the amount of electromagnetic radiation reflected by a body to the amount incident upon it, commonly expressed as a percentage

Aldehyde—any of a class of organic compounds containing the group R–CHO, intermediate in state of oxidation between primary alcohols and carboxylic acids

Alveolus (pl. *alveoli*)—a small, sac-like dilation at the innermost end of the airway, through whose walls gaseous exchange takes place

Analysis, factorial—a method of evaluating certain definite integrals by the use of gamma functions

Anaplasia (adj. *anaplastic*)—a condition in tumor cells in which normal functional and physical differentiation is lost

Anthracosis—a disease of the lungs caused by inhalation and accumulation of carbon particles

Antirachitic—opposing or preventing the development of rickets

Aphid (aphis)—a small sucking insect; a plant louse

Atelectasis—the collapse of all or part of a lung, with resultant loss of functioning tissue

Attenuation—In physics, any process in which the flux density (or power, amplitude, intensity, illuminance, etc.) of a "parallel beam" of energy decreases with increasing distance from the energy source

Auscultation (adj. *Auscultatory*)—the act of listening for sounds within the body, usually with the use of a stethoscope

Bacillus subtilis—a common microorganism found in soil and water and frequently occurring as a laboratory contaminant; rarely implicated in causing disease

Benzo (a) pyrene—a polycyclic aromatic hydrocarbon which under certain circumstances has been shown to produce cancer

Bifurcation—a site where a single structure divides into two branches

Blistering, osmotic—paint blisters caused by moisture picking up water-soluble salts in its passage through the paint film, resulting in an ideal situation for osmosis which represents a tremendous force formation of blisters

Blowby—the leakage of gas or liquid between a piston and its cylinder during operation

Body, asbestos—a fiber of asbestos surrounded by a deposit of protein material

Bronchiectasis—a chronic dilatation of a bronchial passage

Bronchiole—one of the finer subdivisions of the bronchial tree

Bronchitis—an inflammation of the bronchi, usually manifest clinically by cough and the production of sputum

Bronchitis, chronic—a long-standing inflammation of the bronchi characterized by excessive mucus secretion in the bronchial tree and manifested by a persistent or recurrent productive cough. For the purposes of definition, these symptoms must be present on *most days* for a minimum of *3 months* of the year for at least *2 successive years* (American Thoracic Society)

Bronchoconstriction—a diminution in the size of the lumen of a bronchus

Bronchus (pl. *bronchi*)—one of the larger air passages in the lung

Broth—a liquid medium for the cultivation of microorganisms

Capacity, forced vital (FVC)—the largest amount of gas which can be forcibly expired from the lungs following a maximal inspiration

Capacity, functional residual (FRC)—the volume of gas remaining in the lungs at the resting end-expiratory level

Capacity, total lung (TLC)—the volume of gas contained in the lungs at full inspiration

Capacity, vital—the maximum volume of gas which can be expired from the lungs following a maximum inspiration

Carbonation—conversion into a carbonate (which in many cases refers to one or more members of the calcite, dolomite, and aragonite groups of minerals) impregnation with carbon dioxide

Carcinogen—a substance capable of causing living tissue to become cancerous

Carcinogenesis—the production of cancer

Carcinoma—cancer; malignant growth made up of cells derived from epithelial tissue

Carcinoma, bronchogenic—cancer arising in bronchial tissue of the lung

Carcinoma, squamous cells—cancer developing from squamous epithelial cells

Carcinoma in situ—a neoplastic entity in which tumor cells are present but the invasion of normal tissue has not yet taken place

Cardoivascular—pertaining to the heart and blood vessels

Cercospora beticola—a member of the genus *Cercospora*, which consists of imperfect fungi that are leaf parasites with long slender multi-septate spores

Channel black—carbon black made by impingement of a luminous natural gas flame against an iron plate from which it is scraped at frequent intervals; properties vary widely, but the material has an unusually fine state of subdivision and great surface area

Chloroplast—a specialized body (a plastid) containing chlorophyll in the cytoplasm of plants; the site of photosynthesis and starch formation in plants

Cholinesterase—any one of several enzymes that hydrolyze choline esters, occurring most frequently in nervous tissue and the blood

Cilium (pl. *cilia*)—small, hairlike process attached to a free surface of a cell, capable of rhythmic movement

Clearance—the removal of material from the body or from an organ

Clinker—kiln-fired limestone from which commercial cement is made

Coalescence—in cloud physics, the merging of two water drops into a single larger drop

Coefficient, absorption—the fractional rate at which flux density of radiation decreases by absorption with respect to the thickness of the absorbing medium traversed

Coefficient, extinction—the sum of the absorption coefficient and the scattering coefficient for a medium that both absorbs and scatters radiation

Coefficient, scattering—the fractional rate in the transmission of radiation through a scattering medium (as of light through fog) at which the flux density of radiation decreases by scattering in respect to the thickness of the medium traversed

Coh unit—a measure of light absorption by particles, defined as that quantity of light scattering solids producing an optical density of 0.1

Collagen—a fibrous protein forming the main supportive structure of connective tissue

Collector, cyclonic—a centrifugal fractionator in which a vortex of air throws particles out of a stream, where they collect or stick to the surface of a container

Comminute—to break or crush into small pieces

Concentration—the total mass (usually in micrograms) of the suspended particles contained in a unit volume (usually one cubic meter) at a given temperature and pressure; sometimes, the concentration may be expressed in terms of total *number* of particles in a unit volume (e.g., parts per million); concentration may also be called the "loading" or the "level" of a substance concentration may also pertain to the strength of a solution

Conifer—belonging to the coniferales order, consisting primarily of evergreen trees and shrubs

Consolidation—the process by which a diseased lung passes from an aerated collapsible state to one of an airless solid consistency because of accummulation of exudate

Contrast—in visual range theory, the ratio of the apparent luminance of a target minus that of its background to the apparent luminance of the background

Conurbation—a great aggregation or continuous network of urban communities

Coryza—an acute catarrhal condition of the nasal mucous membrane with profuse discharge from the nostrils

Criteria, air quality—a compilation of the scientific knowledge of the relationship between various concentrations of pollutants in the air and their adverse effects

Cryolite—a mineral fluoride consisting also of sodium and aluminum

Cuticle—a varnish-like layer covering the surface of a leaf

Cytoplasm—the protoplasm of a cell (excluding that of the nucleus)

Dead space, anatomic—that part of the airway occupied by gas which is unavailable (by its location) to take part in oxygen-carbon dioxide exchange through the walls of the alveoli

Dead space, physiologic—the volume of gas within the alveoli which does not participate in the oxygen-carbon dioxide exchange through the walls of the alveoli

Dehydrogenase—any one of various enzymes which accelerate the removal of hydrogen from metabolities and its transfer to other substances, thus playing an important role in biological oxidation-reduction processes

Deliquesce—to dissolve gradually and become liquid by absorbing moisture from the air

Density—the amount of matter per unit volume, usually expressed in grams per cubic centimeter

Density, optical—the degree of opacity of any translucent medium; the common logarithm of the ratio of the initial intensity of light to the intensity of transmitted or reflected light

Desorption—the release of a substance which has been taken into another substance by a physical process or held in concentrated form upon the surface of another substance; the reverse of absorption or adsorption

Desquamate—to cast off epidermis in shreds or scales; to peel off in sheets or scales

Deviation, standard geometric (σg)—a measure of dispersion of values about a geometric mean; the portion of the frequency distribution that is one standard geometric deviation to either side of the geometric mean accounts for 68% of the total samples

Deviation, standard normal—a measure of dispersion of values about a mean value; the square root of the average of the squares of the individual deviations from the mean

Dextran—a water-soluble polymer used therapeutically as a plasma substitute

Diameter, count median (CMD)—the geometric median size of a distribution of particles, based on a numerical count

Diameter, mass median (MMD)—the geometric median size of a distribution of particles, based upon a weight (usually derived from a Stokes' Diameter)

Diameter, Stokes'—the diameter that a unit density particle of spherical shape would have if it behaved the same as the particle being studied

Dichotomous—dividing in succession into pairs; showing a dual arrangement

Distal—furthest or most remote from the median line of the body, from the point of attachment, or from the origin; peripheral (*cf.* proximal)

Diverticulum (pl. *diverticula*)—a pouch or cul-de-sac of a hollow organ

Dosimetry—the accurate measurement and determination of (medicinal) doses

Dyspnea—difficult or labored breathing

Earth, diatomaceous—a chalky material used as a filter aid, an absorbent, a filler, an abrasive, and as thermal insulation

Edema—a condition due to the presence of abnormally large amounts of fluid in the intercellular tissue spaces of the body

Effluent—something that flows out, such as a liquid discharged as a waste

Elution—the process of washing out, or removing with the use of a solvent

Elutriator, fractional—a fractional sampler which removes coarse particles from the air by gravity settlement

Emphysema—a swelling due to the presence of air, usually excess or additional air. The term is usually used to refer to pulmonary emphysema

Emphysema, pulmonary—a condition in which there is overdistension of air spaces and resultant destruction of alveoli and loss of functioning lung tissue

Endocytosis—a condition or disease arising from the inclusion within a cell of material which does not properly belong there

Entomology—zoology dealing only with insects

Epidemiology—a science dealing with the factors involved in the distribution and frequency of a disease process in a population

Epidermis—the outermost layer of skin in animals; any integument

Epiglottis—a plate of cartilage which covers the entrance to the larynx during swallowing, thus preventing food or fluid from entering the windpipe

Epithelium—a closely packed sheet of cells arranged in one or more layers, covering the surface of the body and lining hollow organs

Epithelium, columnar—a type of epithelium composed of tall, prismlike cells

Epithelium, spuamous—a type of epithelium composed of plate-like cells

Ergometer—an instrument which measures work done, e.g., by muscle contraction; a dynamometer

Evaginate—to turn inside out or protrude by eversion

Extrapolate—to project data into an area not known or experienced, and arrive at knowledge based on inferences of continuity of the data

Fibrosis—the development of fibros tissue; sclerosis

Filiform—having the shape of a thread or filament

Floc—something occurring in indefinite masses or aggregates

Fluorosis—a pathologic condition resulting from excessive intake of fluorine

Fluorspar—the mineral fluorite

Flux—a flowing or discharge of fluid; a substance used to promote fusion (as by removing impurities) of metals or minerals; the rate of transfer of fluid, particles, or energy (as radiant energy) across a given surface

Fume—an aerosol formed by the condensation of vapors as they cool

Gastric—pertaining to the stomach

Gastrointestinal—pertaining to the stomach and intestines

Goblet cell—a type of epithelial cell containing mucus and having the shape of a flask or goblet

Gravimetric—of or relating to measurement by weight

Haze—fine dust or salt particles dispersed through a portion of the atmosphere; the particles are so small that they cannot be felt or individually seen with the naked eye, but they diminish horizontal visibility and give the atmosphere a characteristic opalescent appearance that subdues all colors

Hematite—the mineral iron oxide; Fe_2O_3

Histamine—a substance which produces dilatation of capillaries and stimulates gastric secretion, occurring in both animal and vegetable tissues; β-imidazolethylamine

Histogram—a graphical representation using a series of bars

Histology—the study of the anatomy of tissues and their microscopic cellular structure

Hydrocarbon—a compound containing only hydrogen and carbon. This group is subdivided into alicyclic, aliphatic, and aromatic hydrocarbons according to the arrangement of the atoms and the chemical properties of the compounds.

Hygroscopic—readily absorbing and retaining moisture from the atmosphere

Hyperplasia (adj. *hyperplastic*)—an increase in the number of cells in and bulk of a tissue, with retention in normal function and cellular structure

Illumination—the process in which light is brought to some surface or object

Impactor, cascade—an instrument which employs several impactions in series to collect successively smaller sizes of particles

Incidence—the rate at which a certain event or disease occurs

Insolation—the rate at which direct solar radiation (of all wavelengths) is delivered to a unit area of a horizontal surface, usually at or near ground level

Insulation—the prevention of the transfer of energy between two conductors by separation of the conductors with a non-conducting material; or, the non-conducting material itself

Interstitial—pertaining to or situated in the space between cells

In vitro—in a test tube or other artificial environment

In vivo—within a living body

Isotherm—a line on a chart representing changes of volume or pressure under conditions of constant temperature; or lines on a map connecting points having the same temperature

Ketone—any of a class of organic compounds that are characterized by a carbonyl group attached to two carbon atoms, usually contained in hydrocarbon radicals or in a single bivalent radical, similar to aldehydes but less reactive

Lacrimation (lachrymation)—tear formation, especially in excess

Langley—a unit of energy per unit area, commonly employed in radiation theory and equal to one gram-calorie per square centimeter

Larynx—the organ concerned with the production of the voice, situated at the upper end of the trachea

Lesion—an injury or other circumscribed pathologic change in a tissue

Logarithm—a number which represents the power to which a given number must be raised to produce another given number

Lumen—the inner space of a hollow organ or tube

Lycopodium—a genus of club-moss; a powder ("vegetable sulfur") used to prevent the agglutination of pills in a box or as a dusting powder

Lymph—a fluid that is collected from the tissues throughout the body, flows in the lymphatic vessels, and is eventually added to the bloodstream

Lymphoma—any neoplasm developing from lymphatic tissue

Macrophage—a large phagocytic cell found in the connective tissue, especially in areas of inflammation

Malignancy—something which tends to become progressively worse and if unchecked could result in death

Mastoiditis—an inflammation of the skull bone behind the ear

Mean, geometric (M_g)—a measure of central tendency for a log-normal distribution;

the value, in a given set of samples, above which 50% of the values lie

Meatus—a natural body passage, particularly the external opening of a canal

Mesothelioma—a tumor which develops from the lining of a coelomic body cavity

Metaplasia (adj. *metaplastic*)—a change in the cells of a tissue to a form which is not normal for that tissue

Metastable—marked only by a slight margin of stability

Meteorological range (*standard visibility, standard visual range*)—an empirically consistent measure of the visual range of a target; a concept developed to eliminate from consideration the threshold contrast and adaptation luminance, both of which vary from observer to observer

Morbidity—the occurrence of a disease state

Morphology—a branch of biology dealing with the structure and form of living organisms

Mortality—the ratio of the total number of deaths to the total population, or the ratio of the number of deaths from a given disease to the total number of people having that disease

Motion, Brownian—the rapid random motion of small particles due to bombardment by surrounding molecules which are in thermal motion

Mucin—a glycoprotein or mucopolysaccharide secreted by mucous glandular cells

Mucopurulent—pertaining to an exudate (or sputum) that is chiefly purulent (pus) but also contains significant amounts of mucus

Mucoviscidosis—cystic fibrosis

Mucus (adj. *mucous*)—the clear viscid secretion of a mucous membrane

Mural—pertaining to the wall of a cavity

Mustard gas—an irritating and toxic volatile liquid used as a weapon in World War I; dichloroethyl sulfide

Naris (pl. *nares*)—a nostril or other opening into the nasal cavity

Nasopharynx—the part of the pharynx (throat) lying above the level of the soft palate

Nebulize—to reduce to a fine spray

Necrosis—localized death of cells

Neoplasm—any abnormal growth, such as a tumor

Nephelometer—a photometric instrument for the determination of the amount of light transmitted or scattered by a suspension of particles

Node—a circumscribed swelling

Node, lymph—one of many accumulations of lymphatic tissue situated throughout the body

Nomogram—a graph that enables one to read off the value of a dependent variable with the use of a straightedge, when the values of two or more independent variables are known

Nucleation—the process of particle growth through collection around a nucleus

Nucleus (condensation nucleus)—a particle in the size range from 0.1μ to 1μ which serves as a nidus on which water or other vapors in the air can condense to form liquid droplets

Olefin—a class of unsaturated aliphatic hydrocarbons of the general formula C_nH_{2n}

Olfactory—pertaining to the sense of smell

Ophthalmic—pertaining to the eye

Otitis—an inflammation of the ear

Palisade tissue—a layer of columnar cells rich in chloroplasts found beneath the upper epidermis of foliage leaves

Parameter—an arbitrary constant which characterizes a mathematical expression

Parenchyma—the specific or functional tissue of a gland or organ, as opposed to its supporting framework

Particle—any dispersed matter, solid or liquid, in which the individual aggregates are larger than single small molecules (about 0.0002μ in diameter), but smaller than about 500μ in diameter

Particulate—existing in the form of minute separate particles

Pathogenesis—the production or the mode of origin and development of a disease condition

Pathology—the study of the essential nature of disease, particularly with respect to the structural and functional changes in organs and tissues

Peritoneum—the membrane lining the abdominal cavity and investing the viscera

Phagocyte—a cell that has the power of ingesting microorganisms and other small particles

Phagocytosis—the ingestion of a microorganism or other small particle by a phagocyte

Pharynx—the upper expanded portion of the alimentary canal lying between the mouth, the nasal cavities, and the beginning of the esophagus; the throat

Photometer—an instrument for measuring luminous intensity, luminous flux, illumination, or brightness by comparison of two unequal lights from different sources

Photomicrograph—a photograph of a magnified image of a small object

Photosynthesis—the formation of carbohydrate from carbon dioxide and water in the presence of chlorophyll and light, in plant tissues

Physiology—a science which studies the function of a living organism or its parts

Phytotoxic—harmful to plant materials

Plasmolysis—the shrinking of the cytoplasm away from the wall of a living cell due to water loss by osmotic action

Plethysmograph—an apparatus for the determination and recording of a change in the size of an organ or limb or body

Pneumococcus (pl. *pneumococci*)—a bacterial organism which most often infects the lung and is a common cause of pneumonia; *Diplococcus pneumoniae*

Pneumoconiosis—a fibrous reaction in the lungs, caused by the retention of certain inhaled dusts in the lungs

Pneumonitis—a general term for inflammation of the lung

Pneumotachygraph—an instrument used to determine the force and velocity of respired air

Polystyrene—a clear, colorless polymer of styrene, an unsaturated hydrocarbon of the form $C_6H_5CH=CH_2$

Potentiation—synergism, as between two agents which together have a greater effect than the sum of their effects when acting separately

Precipitation—any or all of the forms of water particles, whether liquid or solid, that fall from the atmosphere and reach the ground. It is a major class of hydrometeor, but is distinguished from cloud, fog, dew, rime, frost, etc. in that it must "fall," and is also distinguished from cloud and virga in that it must reach the ground

Precipitator, electrostatic—an apparatus for the removal of suspended particles from a *gas* by charging the particles and precipitating them by applying a strong electric field

Predator—an organism living by preying on other organisms

Prevalence—the number of ca[illegible] a disease at a given time

Prodrome—the symptoms prece[illegible] appearance or recognition of an [illegible] disease state

Proteinosis—the accumulation of [illegible] in excess in the tissues

Proteinosis, alveolar—a chronic progressive lung disease characterized by the accumulation of granular proteinaceous material in the alveoli

Proximal—nearest to the center of the body or the point of origin (*cf.* distal)

Radioautograph (autoradiogram)—a radiographic portrayal of an object or organism made by the inherent radioactivity of the object or organism

Rale—an abnormal respiratory sound heard in auscultation of the chest

Ratio, standardized mortality—the ratio of the number of deaths observed in a given population over a given period of time *to* the number of deaths expected to occur in the given population over the same period of time if the given population behaved as any other group of similar composition would during that same period

Reflectometer—a photometric or electronic device for measuring the reflectances of light or other radiant energy

Regression—a trend or shift toward a mean. A *regression curve* or *line* is thus one that best fits a particular set of data according to some principle

Rhinitis—an inflammation of the nasal mucous membrane

Rhinorrhea—"runny nose"

Rhonchus (pl. *rhonchi*)—a dry, coarse sound usually originating from partial obstruction in a bronchial tube

Scattering, Mie—any scattering produced by spherical particles, without specific regard to the comparative size of radiation wavelength or particle diameter

Scattering, Rayleigh—any scattering process produced by spherical particles whose radii are smaller than about one-tenth the wavelength of the scattered radiation

Sigmoid curve—a "bell-shaped" curve serving as a prototype for the normal distribution of data about a mean

Silicosis—a type of pneumoconiosis caused by inhalation of silica dust and characterized by silica-containing nodules of scar tissue in the lung parenchyma

Sorption—the generalized term for the many phenomena commonly included under the terms adsorption and absorption, when the nature of the phenomenon is unknown or indefinite

Spirometer—an instrument for the measurement of the volume of gas respired by the lungs

Spore—a reproductive element of many lower organisms

Squamous—resembling or covered with scales

Standards, air quality—levels of air pollutants which cannot legally be exceeded during a specific time in a specific geographical area

Stasis—the slowing down or cessation of the normal flow

Stigma—the part of the pistil of a flower which receives the pollen granules and on which they germinate

Stokes' Law—a law in physics stating that the force required to move a sphere through a given viscous fluid at a low uniform velocity is directly proportional to the velocity and radius of the sphere

Stoma (pl. *stomata*)—a small opening in the epidermis of a plant

Subcutaneous—beneath the skin

Supercool—to cool below the freezing point without solidification or crystallization

Supersaturation—a condition of containing an excess of some material or force, over the amount required for saturation normally

Synergism—a situation in which the combined action of two or more agents acting together is greater than the sum of the action of these agents separately

Systemic—relating to the body as a whole, rather than to its individual parts

Tannin—any one of a group of soluble astringent complex phenolic substances that are widely distributed in plants

Terpene—any one of a class of isomeric hydrocarbons of the prototype $C_{10}H_{16}$ that are found in many essential oils, but especially from conifers; may also refer to any of various compounds derived from terpene hydrocarbons or closely related to them

Tipburn—a disease of the potato, lettuce, and other cultivated plants, characterized by burning or browning of the tips and margins of the leaflets and caused by loss of water due to excessive heat and sunshine

Toxicology—the study of poisons, including their preparation, identification, physiologic action, and antidotes

Trachea—windpipe; the airway extending from the larynx to the origin of the two mainstem bronchi

Tracheobronchitis—an inflammation of the trachea and bronchi

Turbidity—in meteorology, any condition in the atmosphere which reduces its transparency to radiation, especially to visible radiation

Tyndallometer—an instrument that measures suspended particle concentration by the amount of light scattered out of a beam

Ultrafilterable—capable of being separated by a dense filter which is used for the filtration of a colloidal solution holding back the dispersed particles but not the liquid

Updraft—an upward movement of air or other gas

Ventilation, minute—the total volume of gas respired in one minute, i.e., the tidal volume multiplied by breaths per minute

Visibility—In United States weather observing practice, the greatest distance in a given direction at which it is just possible to see and identify with the unaided eye (a) in the daytime, a prominent dark object against the sky at the horizon, and (b) at night, a known, preferably unfocused, moderately intense light source. After visibilities have been determined around the entire horizon circle, they are resolved into a single value of prevailing visibility for reporting purposes.

Visual range—the distance, under daylight conditions, at which the apparent contrast between the specified type of target and its background becomes just equal to the threshold contrast of an observer; to be distinguished from the night visual range. The visual range is a function of the atmospheric extinction coefficient, the albedo and visual angle of the target, and the observer's threshold contrast at the moment of observation.

Volume, forced expiratory (*FEV*)—the volume of gas forcibly exhaled over a given time interval (usually measured in seconds) after maximum inspiration, e.g., $FEV_{1.0}$ for this measurement over a 1.0 second period.

Volume, minute—same as minute ventilation

Volume, tidal—the volume of gas inspired or expired during each respiratory cycle.

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